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Proceedings

of the

Sixth Annual American Association

of

Equine Practitioners Convention

Phoenix, Arizona
DECEMBER, 1960

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Sixth Annual American Association

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Equine Practitioners Convention

HOTEL WESTWARD HO,
PHOENIX, ARIZONA

December, 12 thru 14, 1960.

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FOREWORD

This is the fifth annual proceedings book of the American Association of Equine Practitioners covering the sixth annual convention held in Phoenix, Arizona, December 12 through 14, 1960.

The proceedings of the business sessions were transcribed and are a matter of record. For purposes of brevity and economy, this book is confined to an edited version of the scientific papers, panels, open discussions and the banquet address.

There is one regrettable omission from this book — the presentation by Dr. C. H. Reid of Hollywood, California. This consisted of an extemporaneously delivered description of a very large number of photographs and slides which, on economic grounds, could not be adequately reproduced in a book such as this. A summary of Dr. Reid's excellent talk will, however, be found in the appropriate section.

Dr. E. E. Leasure, President of the American Veterinary Medical Association, delivered the major address at the annual banquet.

F. J. MILNE, D.V.M.
Editor.

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Photographed at the conclusion of the 1960 A.A.E.P. Convention. From left to right:—Gen. Wayne O. Kester, Drs. M. L. Scott, J. G. Woodcock, M. B. Teigland, W. O. Reed and F. J. Milne.

1960
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BANQUET ADDRESS

by

E. E. Leasure—President A.V.M.A.

It is a real pleasure for me to come to Phoenix as your guest and participate in your sixth annual meeting. I am indeed happy to bring to you as a representative of the A.V.M.A., greetings and best wishes for a very successful and profitable meeting. From the effort that has gone into the planning of your program, and if attendance may be relied upon, then I am sure that there is a genuine interest in equine practice and that your motivation is to be better informed in order to render a better service to your clients, and to your patients.

The area of equine practice is undoubtedly the oldest specialty in the veterinary profession, and indeed, the veterinary profession as we know it today in this country, leaned heavily upon this specialty as foundation upon which to build, yet your organization is perhaps the youngest among a rather sizeable list of specialty groups within the veterinary profession. As your organization has growing pains, and I am sure it will, you will experience many problems. Among these will be constitution and by-law changes particularly with regard to the qualification of applicants for membership. I can foresee the question arising, if indeed it has not already arisen — how will this specialty group pass upon applicants for membership — will it accept all veterinarians who apply or will there be certifying standards set up that all applicants must meet, and if so, what will these be and furthermore, how will they be determined and passed upon? What about continuing education for the Equine Practitioner over and above conferences and conventions? Are any proposals being developed?

These questions are important to a young and growing specialty group and eventually must be answered.

It seems to me that your association should be concerned about the professional proficiency of its membership and to this end, set up the mechanism whereby appropriate certifying standards may be determined, and yet, these should not be so rigid that they cannot be met by the applicant who is willing and able to accept the challenge of improving himself for certification. I wrote these remarks some days ago and last night while attending your executive board meeting, I found my predictions to be true. You are having growing pains and you are thinking about constitution and by-law changes, membership proficiency and education.

Veterinary medicine as you are all aware, is a constantly growing and changing profession, and this is as it should be. Many factors are responsible including those of advancements in education, agricultural and veterinary economics, diminishing

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and/or increasing animal populations among certain species, a changing livestock and poultry production, and the needs of the public for veterinary services.

One of the greatest of these changes occurring during the last 50 years, was in the area of equine practice, and if you'll pardon this personal reference, I am of an age that permitted me to observe and participate in the changing pattern. I was born in the horse and buggy age, during the latter part of which the horse and mule population of America exceeded 30 million and has since decreased to a low of 3,078,000 in the United States during the current year. The increase of the pleasure horse now almost matches the decrease of the work animal.

The rapidly decreasing horse population following the year 1910, undoubtedly contributed to the diminishing number of individuals entering the veterinary profession in the next 15 years. At the end of this period, 1925, existing veterinary schools experienced their lowest enrollment.

I remember well the great Kansas horse plague outbreak of the 1911-1913 period, when thousands of horses were infected and died. The best veterinary talent of the day was rushed to the scene; these veterinarians were perplexed for they did not understand the nature or the cause of the disease. Many theorized that they were dealing with a central nervous infection and research projects were instigated, but it was years later before the true cause, a specific virus, was established. Treatment of these striken animals was for the most part empirical and symptomatic, and even in some cases, inhumane. The importance of good nursing was recognized and perhaps accounted for as many recoveries as all other treatments put together. I recall the futile attempts of our local veterinarians with their eserine sulphate and arecoline hydrobromide injections, their aloes boluses, their oil and magnesium sulphate drenches, and their around-the-clock hot and cold kidney packs in attempting to provoke a cure. I further recall some of the lay and quack treatments, one of which was hot olive oil poured into the ears, for there were those who believed a small parasite in the external canal of the ear was the cause of the disease; and another, the so-called shock treatment.

I recall also, subsequent outbreaks in the 1920's and 1930's, and that our knowledge of the disease had increased many fold, that its etiology was determined, and a good prophylactic vaccine was found, thanks to the research efforts of our laboratory colleagues.

My foregoing remarks were intended to document the fact that veterinary medicine is a constantly changing profession and that these changes must be met as they arise. I could have cited just as well, the great revolution of the poultry industry and its effect upon the profession, and the more recent trends in swine and cattle production — so-called "Vertical Integration" — that

is having and will have a tremendous impact upon the veterinary profession.

During these last 50 years the veterinary profession through education and research, experience, trial and error, has increased its store of information about the horse, its diseases, surgery, treatment, and management, many, many fold. Fallacies of yesterday need not be fallacies of today. Yet we find among us and among our clients, those who fail to take advantage of the available knowledge and information at hand, and those who prefer to cling to some silly superstition that has been handed down from generation to generation.

In preparing this address, I became curious as to what was appearing in the veterinary literature relative to articles on the horse so I thumbed through the two A.V.M.A. journals, commencing with the issues of January 1950.

Although I hesitate to vouch for the complete accuracy of my statements, I found that the Journal of the A.V.M.A. carried 167 articles through December 1958, and the Research Journal carried 36 research articles through the same period, for a grand total of 2:03 articles. I would suspect that these would represent 50% or slightly more of all articles on the horse appearing in all veterinary publications in America.

While this rather shallow and incomplete survey indicates that 50 or more articles are appearing yearly, I am not too encouraged. I believe we need to increase the volume many times. In this literary search also, I thought it would be interesting to know when the last good textbook on diseases of the horse appeared. Frankly, my friends, I would be ashamed to tell you. It is indeed hard to understand why more is not written about this important area of the veterinary profession.

This lack of a recent book on the diseases, treatment and surgery of the horse offers a real challenge to the American Association of Equine Practitioners. I am certain that there is plenty of talent in the profession to assemble a comprehensive book.

Were it not for the fact that a great percentage of the available knowledge of the horse, relative to diseases and treatment, physiology, pathology, surgery, and management is incorporated in various texts, veterinary schools would find it exceedingly difficult to do justice to this phase of teaching.

It is easy for anyone to make suggestions and recommendations to another for development in his specific area, but the matter of suggesting how they can be implemented is quite another thing.

After reviewing the research titles mentioned previously, and interviewing a number of practitioners in the field, I am convinced that solving many of your problems will be accomplished only through research.

This matter of developing more research activity in the

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overall area of equine problems becomes one of your organization's responsibilities. Your membership should be fully indoctrinated as to the need and value of research and stimulated to fully support it. I am wondering if any thought has been given to the need for establishing an Equine Research Institute. Although the process of research may be slow and unrewarding at times, there are many ways to stimulate more to the done.

I understand that some of the pertinent problems confronting the equine practitioner and which require investigation are:

(a) Acute hemorrhagic gastro-enteritis
(b) Sterility in the mare
(c) Distemper-strangles complex
(d) Parasite eradication and control
(e) Infectious equine anemia
(f) Diseases of the young foal
(g) Nutrition and management
(h) Improved surgical procedures
(i) Lameness
(j) Others.

This is a very short list and I am sure that all of you can add many more. It is indeed a rare accomplishment to solve completely an animal disease complex and while we may learn to live with diseases, we nevertheless must continue our efforts toward complete solution. Some of the foregoing problems have been with us almost as long as the horse. Considerable information is available, but do we have the strongyle problem whipped and how about strangles, infectious anemia, sterility, and others? As education in the profession progresses we will be faced with new entries and we must be ready to face these new challenges.

I have been informed that your organization, since its first convention, has been quite well indoctrinated in Organized Veterinary Medicine by several Past Presidents of the A.V.M.A., therefore, I shall refrain from such remarks. However, I should like to mention some of the activities that the A.V.M.A. is engaged in, and particularly some of the most recent.

Many of you attended the 97th Annual Convention in Denver last August, and know first hand, what a wonderful meeting it was. The proceedings of this convention will soon be off the press; they will be easier to read than formerly, and I would like to recommend that you all take the time to read them in order that you may be better informed about the activities of the A.V.M.A.

Since last September, your elected officers and members of the Headquarters Staff have had a heavy travel schedule attending constituent association and allied professional meetings and attending to the business affairs of the A.V.M.A.

The various councils have met in Chicago and elsewhere, and their reports will appear in forthcoming issues of the Journal.

The A.V.M.A. Executive Board and the Board of Governors met in November to discuss and administer the association's business. Some of the items on which action was taken and worthy of passing on to you: a suitable working plan for handling the Women's Auxiliary office matters by A.V.M.A. staff members was adopted; plans were made and discussed for expanding the public relations and professional relations departments of the A.V.M.A. The Executive Board made a great, and perhaps historical, decision when it approved an expenditure of up to $25,000 to launch the first phase of the Prospectus Survey.

The Prospectus Committee visualizes this very broad and comprehensive survey of veterinary medicine in North America to be conducted in two phases. The first phase, which will take about a year, is to employ a professional agency to determine what the association wants, what acres of the profession are to be surveyed and how the survey will be conducted. The second phase will be the actual conducting of the survey by a professional agency. When completed in two or three years, we should have an up-to-date, accurate appraisal of the status of our profession, wherein our strength lies, our weaknesses, and how we should proceed in the future.

It is presumed that after the first phase is completed, the three or four hundred thousand dollars needed for the second phase of the survey can be obtained from agencies outside the profession. I consider this to be the most important task facing the profession today, and when completed, will guide us in the future.

The second annual Constituent Secretaries Association meeting was held in Chicago in November; over three-fourths of the associations were represented and from all reports, the meeting was a grand success. At this meeting there was a free exchange of information that will enable all associations to build a better organized veterinary profession.

The Board of Governors met again in December, to interview applicants to head the Public Relations Department. They employed an individual, presently, Acting Director of the American Dental Association's Bureau of Public Information, operating out of Chicago. He has had a broad experience in radio and television communications, in insurance, as a freelance writer, and with the Dental Association. For the past year he has directed the Dental Association's nation-wide public information and publicity program. He is young and vigorous and should bring to the A.V.M.A. the kind of service and leadership it has been seeking. He comes to us very highly recommended and will take office on January 1, 1961.

You probably are all aware of the A.V.M.A. Research Trust Fund. This fund was established to assist eligible veterinarians to pursue advanced education in teaching or research in any of

the various disciplines of veterinary medicine. The fund provides fellowship grants to qualified individuals which will enable them to keep body and soul together while engaged in graduate study at any veterinary school in America. Early last winter, the Council on Research, charged with the responsibility of administering the fund, realized that there were barely enough funds to continue the program through this current year and next year. Subsequently, the A.V.M.A. solicited the aid of the Women's Auxiliary to initiate a nation-wide drive for $75,000 to augment this program. The Auxiliary accepted this challenge, and cooperating with the A.V.M.A. staff, have worked out elaborate plans to launch their drive on February 1, 1961. Every veterinarian in the U. S. and Canada will be contacted personally. Won't you cooperate with the Auxiliary in this highly worthy cause?

For the past several years, educators in the medical sciences and other professions have been worrying about the declining numbers and the declining quality of applicants seeking admission to educational institutions, and veterinary medicine was no exception. Therefore, last year, the A.V.M.A. quietly launched a campaign to recruit the superior high school student for a career in veterinary medicine. Constituent associations and auxiliaries were requested to assist in placing the A.V.M.A. brochure entitled "Veterinary Medicine as a Career" in every high school library of America; and individually into the hands of every interested student. Veterinary practitioners were encouraged to inform superior high school students of their acquaintance to consider a career in veterinary medicine. The program is going forward this year with new innovations being planned. The profession needs the superior student just as much as the other sciences and professions need them, and we must put forth every effort to obtain them. That the program is bearing results is already evident because there was a nation-wide increase of enrollment in the pre-veterinary curriculum last September, although not great enough to evaluate as significant.

In closing, I realize that I have rambled over more than one subject, but since we are all veterinarians, they seem to me to be pertinent.

I am happy that an intense interest in equine practice has been revived and I hope it will continue to grow.

In summary, may I suggest that you consider these items:

(a) That you put your house of today in order for the future tomorrow,

(b) that you stimulate and support all phases of animal disease research, and

(c) that you support the educational recruitment program of the A.V.M.A. in its quest for superior students and in its program of graduate education.

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EQUALIZERS FOR EQUINE PRACTICE

E. R. *Walker*

Every one of us in this room has an overabundance of left-handed days. I think this is a fact with which no one will argue. Naturally, we would like to do away with as many as possible. In an attempt to do this, the members of our clinic have used everything from standardizers to tranquillizers.

A group of preparations, instruments and techniques has made our work far less tedious. We refer to them as "equalizers". I would like to show you slides of these and discuss them, in the hope that they will help you with your problems.

[Slide]. This product is a broad-spectrum mastitis ointment, containing dihydrostreptomycin, penicillin, bacitracin, and papain. We use it on necrotic ulcers and fistulous tracts. By using this on areas once a day for three days, we find that we will get rid of many of these troublesome areas.

[Slide]. I am sure you are familiar with this product, Varizyme. It has so many uses that it is almost a shotgun-type product. We use 125,000 units intravenously once a day for three or five days. We have also used it in conjunction with neomycin and cortisone in cases of acute tendinitis in range-type horses. It eliminates much of the hot packing and tedious labor which we previously carried out. In the last month we have had the opportunity to use Varizyme on two cases of concussion, where the horse had reared over backwards. We started treatment the fifth day after the concussion occurred, feeling that by then, the seepage of blood would have stopped. By the time that we had administered the fifth dose, the horse was walking normally.

[Slide]. This multiple-use product is a pink wound dressing consisting of scarlet oil, zinc oxide, fish oil, DDT and raw linseed oil. We like it especially because we treat many half-broken ranch horses. Every third day we apply it to long tears and neglected wounds, such as occur on the rib cage, over the rump, and on the pectoral muscles. One of my good clients says the reason why we sell so much of this is that it has such a passionate pink color!

[Slide]. We use this product, 2% procaine hydrochloride, in cases of impaction in range-type horses by giving 80 cc intravenously as rapidly as possible. There is only one precaution: warn the owner not to plan on riding the horse home because the reaction is very violent. We certainly do not recommend it for use on thoroughbreds. In favorable cases defecation will occur within

thirty minutes. It is a very good prognosticator, in that a negative response to the injection will indicate a serious type of impaction.

Necropsy of such impactions reveals that these horses usually have a torsion of the intestinal tract or intussusception, so that it would not have made any difference what you did. It is much easier to use this product because you do not have to pass the stomach tube, and you get miraculous results.

[Slide]. This is a dental anesthetic called "Carpule". We would not do without it. It is available through any Winthrop depot, and contains novocaine, 2 per cent; pontocaine hydrochloride, 0.15 per cent; cobefrin 1:10000. We appreciate its consistent effect; in other words, it does not seem to vary from horse to horse. It will give anesthesia in 90 seconds, and lasts as long as four to five hours. We like it especially for firing horses, in that, following infiltration, there is no tissue reaction and the owner is usually home with the horse before the anesthetic wears off. We also find it useful for blocking nerves for the diagnosis of lameness and for topical anesthesia of the eye.

[Slide]. Several years ago we started diluting phenothiazine suspension. We have certainly wormed a lot more horses and have obtained better results. We place 20 ounces of phenothiazine suspension (12.5 grams per ounce) in a gallon jug and add enough water to make a gallon. The reasons we feel we obtain better results are: 1) this mixture flows down the stomach tube more easily; 2) it is dispersed through a wider area, due to the added volume; 3) going out and seeing a sick horse off his feed about the third day has practically been forgotten.

After we have mixed, we give 1 pint of the stock solution which contains 30 grams of phenothiazine per pint. This is accepted as the dose for a 1000-pound horse. A yearling (600-800 lbs.) receives two-thirds of a pint.

[Slide]. Since some of the horses would not eat the phenothiazine powder available on the market, we devised a formula containing 52 per cent phenothiazine, 27 per cent carob flour, oil of anise and a sweetening agent. We administer this routinely to brood mares, giving 1 tablespoon once a day for four consecutive days in a month. Yearlings receive one teaspoonful in the same manner. It acts as a very good control agent. In horses that are heavily infested with strongyles, we find that we are better off to administer this for four days, wait two weeks, and give it again for four days. A therapeutic dose of worm medicine is given in 2 weeks. Because we do not get such a violent reaction, we feel that this routine has probably done more to control worms than the older method of a twice yearly treatment.

[Slide]. These are plastic stomach tubes. We use them in the winter when the koroseal tube gets stiff. The tube on the left is used on colts, the one on the right on larger horses. They are available from the Harshaw Chemical Company in Houston, Texas. Following cold sterilization, we put them in these No. 20 nail bags and staple the top of the bags. We carry four or five of them to the race track or to a brood farm. When we break open the bag, the owner knows that the tubes will not transmit disease to his animals.

[Slide]. This is a Bayer's dental wedge available through Diamond Laboratories. It is not cumbersome and there is not as much danger of getting whacked across the head with it by a fractious horse. It is simple to insert and can be held between the teeth by tying the handle to the headstall with a piece of cord. By merely inverting it, we have a gag that will work on the other side of the horse's mouth.

[Slide]. For vaccinating horses for encephalomyelitis, we use a tuberculin syringe with ¼ in. I.D. needle. With horses not accustomed to much handling, you can merely slide the hand up along the side of the neck, insert the needle, and it will not go into the subcutaneous tissue. It saves a lot of handling.

[Slide]. This is a gas-propelled wound dressing dispenser which can be adjusted to emit a light or heavy spray. It is especially useful for treating chronic wounds on animals which object to much handling. Depending upon your markmanship, you can hit anything up to 15 feet! This instrument is available from the Quickfield Corporation, 1937 Edmonson Street, Houston, Texas.

[Slide]. This is an aluminum splint that we use for the treatment of wire cuts on the posterior surface of the foot. The slide shows four or five of these splints. You can add to them, if you have a heavier horse; you can subtract if you have a lighter horse. They are very easy to bend. We put them on the anterior surface of the foot and wrap a plaster of paris bandage around the limb, while leaving a window through which the wound is treated. You will notice the flanges at the top and bottom of the splints. They keep the plaster of paris from slipping up or down the limb.

[Slide]. This is a dog catheter we use for flushing out the nasolacrimal duct. It is pliable and will not tear out the ventral end of the duct. Secondly, it gives you a lot of running room out in front of the horse, if you have an unruly horse!

[Slide]. I am sure you are all familiar with this type of wound, the long neglected tear that, up to two or three years ago, there was not much that we as members of the clinic could do, except to debride the tissues and tell the owner to take the animal home and treat the tear as an open wound.

[Slide]. This is not a fish story, as some of you may think but the suture material we use for the type of wound just shown is Ashaway Catfish line, available at most hardware stores. It is a corded line, with a lead cord. It has several advantages in that once the knots are tied, they stay tied. Secondly, it causes little or no tissue reaction. Thirdly, it can be crimped after threading the needle, therefore will not slip out of the needle.

[Slide]. In treating these long wounds, we first find that sterile bandage will remove more of the extraneous material from the wound than will a piece of cotton. A human drain in which four or five holes have been cut is placed in the wound and anchored with a piece of catgut. If necessary, antibiotics can be placed in the end of the drain. The drain is removed by the owner, three to five days later (Fig. 1).

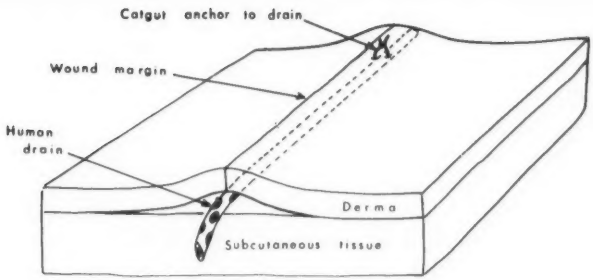
[Slide]. After placing the drain in the wound, double strands of Ashaway catfish line are inserted in the manner of a routine interrupted suture (Fig. 1 Step 1).

[Slide]. The suture material is tied over lengths of intravenous tubing which act as quills (Fig. 1 Step 2). This is very similar to the old button type of suture. However, in this case the rubber quill lends itself better to the contour of the body and healing will occur with much less scarring.

[Slide]. Finally, the ends of the suture are tied over the top of the wound (Fig. 1 Step 2). This gives us three areas of very evenly distributed tension. In the illustration, it looks as if the knots are in contact with the skin incision, but actually the presence of the intravenous tubing will prevent pressure on the incision line (Fig. 1 Step 4).

[Slide]. This is another type of wound in which we use this technic — a lacerated incision above the fetlock. In this particular filly, we sutured the wound and removed the sutures two weeks later.

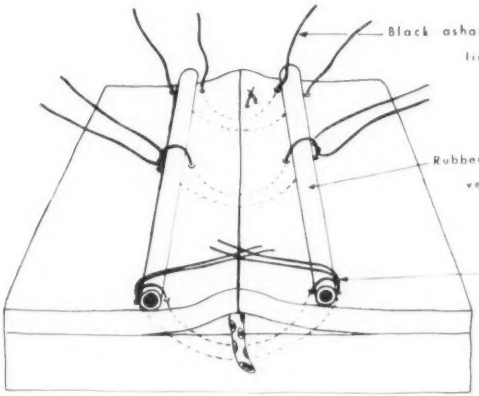
That concludes my presentation. As the old saying goes, if the shoe fits you, wear it. I hope you all have found a pair that will fit you.



STEP 1

STEP 2

STEP 3



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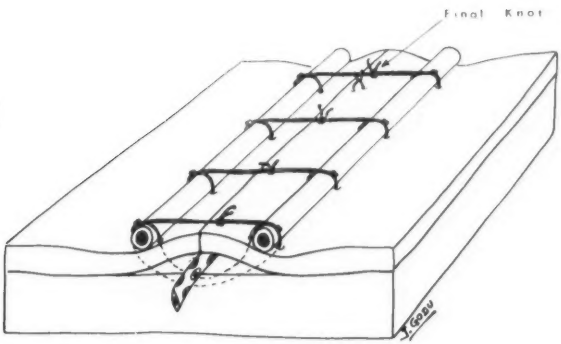

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SUBCUTANEOUSLY INDUCED COUNTER-IRRITATION*

F. J. MILNE

Counter-irritation is a procedure in which certain drugs or other agents are applied to the intact skin in order to cure or ameliorate a deeper situated pathological process. Agents which cause counter-irritation are known as counter-irritants and include the actual cautery, (commonly referred to as firing) acid firing, setons, rubefacients, vesicants and pustulants, the latter two being referred to as blisters. The subcutaneous injection of certain agents — the so-called inward blisters — also merits inclusion in a study of counter-irritation.

Counter-irritation as a form of therapy in human and veterinary medicine has been known and used at least since the Neolithic period (7000-3000 BC). It is interesting to note, however, that despite the fact that this mode of treatment has been in use for so long, the exact way in which it acts and brings about results is not known. Although the more severe forms of inducing counter-irritation have long been abandoned by the medical profession, they are or were until the advent of certain anti-inflammatory compounds, still employed by many veterinarians in equine practice for reasons which to the pathologist and physiologist are quite irrational. The forms of severe counter-irritation just referred to are, as you might have expected, firing and blistering. Since this is not a dissertation on the pros and cons of such therapy we will detour around that argument and go on later to a consideration of subcutaneously induced counter-irritants.

A review of the literature on counter-irritation is a phenomenal task as I found to my cost when preparing a thesis on this subject for a post-graduate degree. Most of the material was written in languages other than English and practically all of it referred to the use of firing and blistering. Few references were found to acid-firing and even less to the use of "inward blisters". Time does not permit a consideration of the literature but if any of you wish to study this topic of counter-irritation to a much greater extent and detail than is present in our text books I shall be happy to contribute my reference list to you with the proviso that you arrange for your own translations from the German, French, Danish, Portugese, Italian, Hugarian and Russian languages!

*Part of a thesis entitled "Thoughts and Observations on Counter-irritation in the Horse" presented before the Faculty of the University of Zurich, Switzerland, July, 1960.

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Some years prior to the study of the effect of "inward blisters", I had carried out a series of observations on the effects of perforating firing on the anterior aspect of the large metacarpal bone and also on the flexor tendon region of normal horses. We also confirmed that the temperature of the firing iron at the conventional cherry-red coloration was in the region of 650-750 degrees Centigrade. The conclusions reached were as follows:—

1. The periosteum and underying metacarpal bone were quite unaffected following perforating firing as commonly carried out in North America.

2. Only rarely was there any evidence of the iron having penetrated deeper than the fascia surrounding the superficial flexor tendon. In other words, using the accepted method of firing in North America, the term perforating firing is virtually a misnomer, as far as deep-seated structures are concerned.

3. The reason for these two conclusions lies in the fact that the rate of heat dispersal from the iron at 650-750 degrees is so great that by the time perforation of the skin is achieved, insufficient heat remains to exert an advantageous effect on the more deeply situated structures. This would tend to imply that if the deeper tissues were in any way involved as a consequence of the firing procedure, then the result of firing was not due to the direct application of heat to the tissue actually requiring treatment, but to the hyperemic effect mentioned by some earlier workers or to other factors as yet unknown.

This theory of heat dispersal was confirmed by carrying out temperature recordings (employing a thermocouple and potentiometer) on living anesthetized horses being subjected to the firing iron. After completing this work we learned that similar results had been obtained by an Italian worker (Bossi) fifty years earlier! Fortunately our results were in accord with those recorded in Italy.

Observations, both gross and microscopic, had also been carried out on the effects of two commercially prepared blistering agents and the familiar red iodide of mercury blistering ointment. A research worker is not supposed to anticipate an effect but the results of employing these agents were surprisingly negative as far as the deeper tissues were concerned. In many cases, actual vesication was not achieved and where vesiculation and pustule formation was noted, the epidermis was largely destroyed, but beyond the subcutaneous tissue there was little and in many cases no evidence of an inflammatory reaction. By far the best results, topically speaking, were obtained by the intermittent but

regular use of a proprietory preparation called Capsolin (Parke, Davis & Co.) and this agent has been used as a substitute for the firing iron for the past 8 or 9 years, by this writer. It is not classified as a blistering agent, although it may, by intensifying the application, act as a vesicant.

Consequent on these somewhat negative results when using accepted methods of counter-irritation, it was decided to observe the effects of certain agents when employed as "inward blisters" and to determine if any deep hyperemic effect could be attributed to their use. Although this type of therapy is familiar to most equine practitioners, a literature search failed to reveal any study of its effects on soft tissues. Perhaps the first known such agent is Oil of Turpentine which when injected subcutaneously will produce a sterile abscess with an accompanying severe inflammatory reaction. The severity of the reaction lead to the dropping of this form of treatment from the veterinarian's armamentarium.

On writing to one firm which produces an inward blistering agent in this country, asking for details about their product, their reply showed great interest in me as a potential buyer but that interest soon evaporated when I explained that it would be necessary for me to know the active principle in their formula if comparative effects were to be carried out. I received no further word from this firm and was therefore left to draw my own conclusions.

Of the agents employed in this study, greatest accent was placed on the use of Lugol's Iodine in dilutions varying from 1 in 20 to 1 in 2, and only the effects of this agent will be discussed at this time. In all cases the back tendon region of the left fore leg was treated and the right fore leg acted as control. All horses utilized were healthy, not lame, and showed no evident limb pathology on clinical examination. Each horse was treated only once, the amount of solution and the number of injections varied considerably. To minimize resentment to the injections, a 25 gauge needle was employed. The animals were killed at varying periods after treatment and macroscopic and microscopic examination of the tissues was carried out.

The following conclusions were reached:—

1. Despite the use of a small bore needle, each injection was felt by the horse — not at the time of the needle puncture but immediately after deposition of the agent in the tissues.

2. Hyperemia could be induced in any tissue in which it was possible to inject the solution. The degree of hyperemia varied directly with the strength of the iodine, the stronger solutions producing evidence of disseminated areas of necrosis on histological examination.

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3. A non-painful edema of the metacarpal region was an invariable finding. The horse never resented palpation of the treated area. If the horse was left alone in the stall, the edema would organise and create a firm filling of the area but if daily walking exercise was given the edema would completely disappear within 40 - 60 days of treatment.

4. Histologically the reaction in depth which these injections stimulated, was infinitely greater than that obtained following the usual firing and blistering techniques, and this was achieved without any pain or upset to the horse, excluding of couse, the momentary stinging effect following injection. The induced active hyperemia is thought to be preferable to the passive hyperemia brought on by methods which induce a prolonged painful response as is the case with the older methods of counter-irritation.

To repeat the necessary detailed statistics from a thesis would surely cause a soporific but not a tranquillizing effect on any audience and since it has been said that a picture is worth a thousand words, I now propose to show some slides and in so doing omit the utterance of several thousand words.

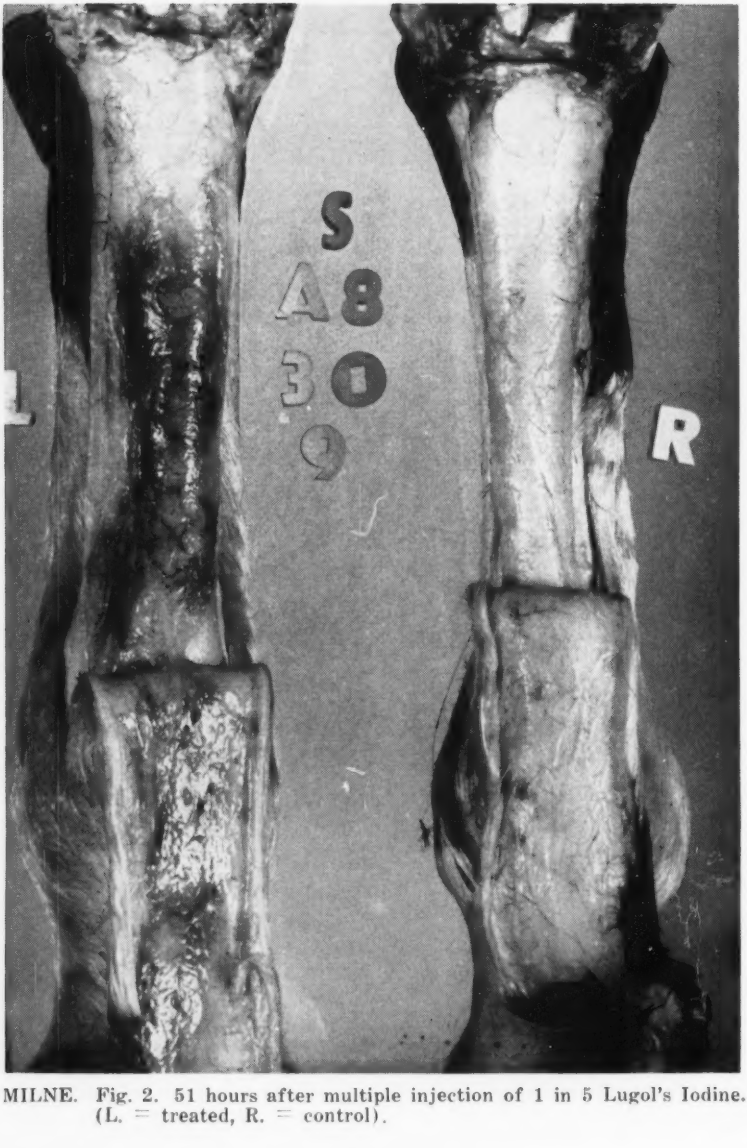
EDITOR'S NOTE:

At this stage of the proceedings the speaker showed a series of colored slides illustrating the effects of the injections on the tissues. Also included were slides on the effects of perforating firing, acid firing and the use of Capsolin ointment. For economic reasons it was not possible to reproduce these slides in color, but a few have been reproduced in black and white.

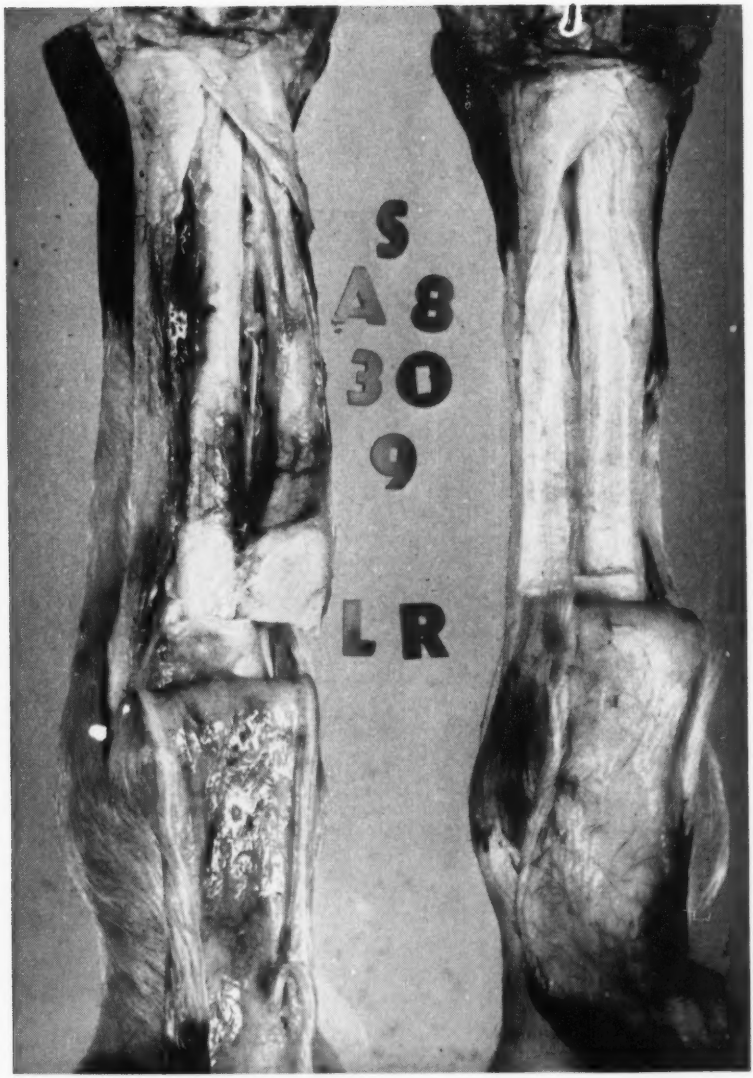
If we accept the often postulated belief that the beneficial effects of counter-irritation are due to the hyperemia induced in the tissues, then the question immediately arises — Why did such methods as the use of ultrasonics and diathermy not live up to expectations? The answer would appear to be contained in the words "tissue destruction". Where tissues are destroyed, scar tissue formation repairs the breach. This may explain why inward blisters and penetrating firing by the older-fashioned irons gave beneficial results, however scar tissue at best is in many cases a rather inadequate substitute for the original tissue and as Thorndike points out there may remain a weak spot which no amount of scar tissue can compensate for. These then are the cases which will not show a beneficial response to any form of therapy.

In conclusion, if by carrying out any form of counter-irritation we wish to produce deep hyperemia and perhaps even some tissue destruction, then the colored slides just shown, demonstrate I suggest, a non-painful and yet highly effective method of achieving our objective.

MILNE. Fig. 1. 72 hours after injection of 1 in 5 Lugol's Iodine lateral to the tendons. Note the extent and depth of reaction between the deep flexor tendons and the suspensory ligament.



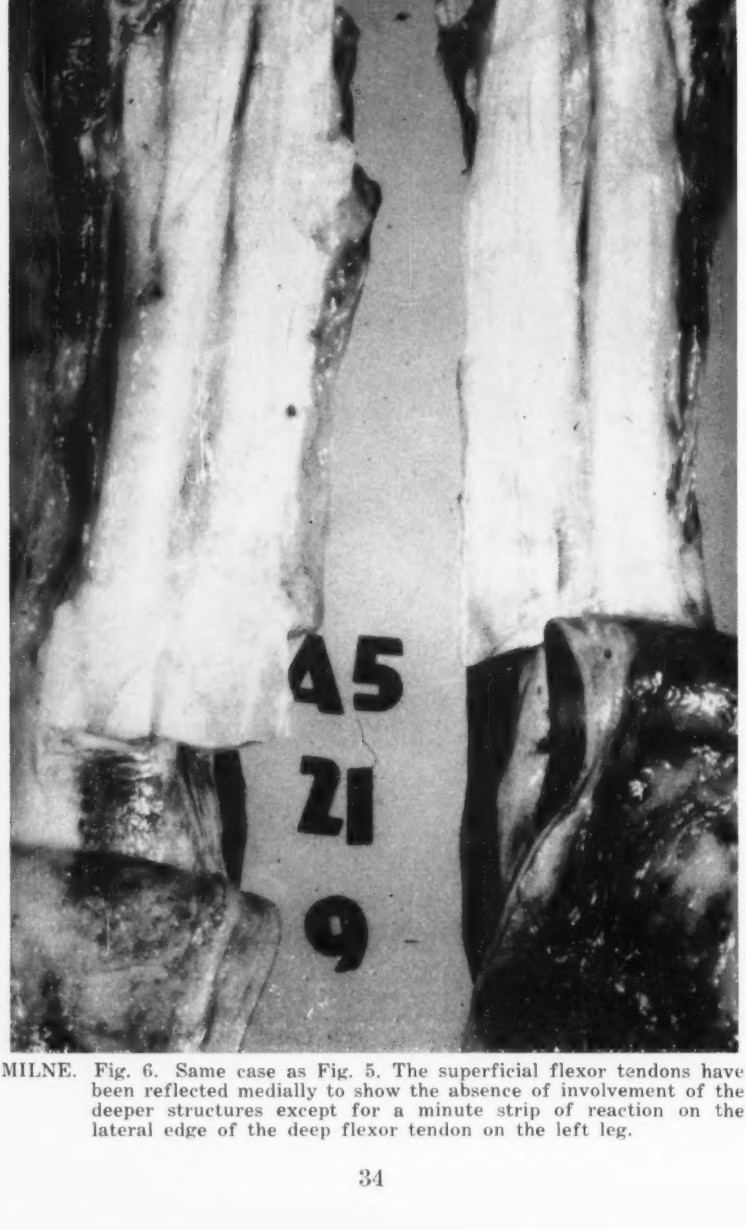
MILNE. Fig. 2. 51 hours after multiple injection of 1 in 5 Lugol's Iodine. (L. = treated, R. = control).



MILNE. Fig. 3. Same case as Fig. 2. Note the reaction between the superficial and deep flexor tendons.

MILNE. Fig. 4. Regressing inflammatory reaction 31 days after multiple injection of 1 in 20 Lugol's Iodine. (L. = treated, R. = control).

MILNE. Fig. 5. 71 hours after application of firing and leg paint to left leg and firing alone to the right leg. Note the favorable comparison.



MILNE. Fig. 6. Same case as Fig. 5. The superficial flexor tendons have been reflected medially to show the absence of involvement of the deeper structures except for a minute strip of reaction on the lateral edge of the deep flexor tendon on the left leg.

PANEL ON EQUINE RADIOLOGY

Moderator: L. E. Johnson

Panel: M. A. Thom
J. B. Chassels
D. L. Proctor
C. F. Reid

MODERATOR JOHNSON: This is to be a panel on radiation treatment for lameness in the horse. Dr. Myron Thom, one of the pioneers in the use of radiation in equine practice, is our first speaker and will discuss acute inflammation and the indications for radiotherapy.

DR. THOM: We go through cycles in any specialty, for instance, in the thirties and early forties, radiation therapy was considered an excellent treatment for many acute inflammatory conditions and certain types of infection. The first major installation for veterinary radiotherapy in this country was, I believe, at the University of Pennsylvania where Dr. Emmerson set out primarily to treat actinomycosis but revised his intention and intended to emulate the work that had been carried out at the Pommer Institute in Vienna, — the treatment of pneumonia in horses. About the time the machine was installed, sulfonamides and then antibiotics came into vogue. Then along came the corticosteroids and the atom bomb and the scare about using radiation for anything. Since then, this particular field of radiation has been somewhat neglected.

In any acute injury, whether it is a bowed tendon or the pulling loose of a tendon insertion as in peritendinitis/periostitis, hyperemia results. The area fills with blood following which there is a temporary stasis of blood flow. Because the blood is moving more slowly in the area, the leucocytes tend to marginate and by ameboid movement they pass through the stretched walls of the blood vessels into the surrounding tissues. The whole area becomes swollen and full of blood cells. This is the stage of acute inflammation and, in the opinion of many radiologists, the time to use radiation. The reason for this is very simple. Of all the cells in the body, the leucocytes are the most sensitive to radiation. They are destroyed more quickly than any other body cells and with their destruction there is a diminution in swelling and also a release of certain antibodies, the enzymes which aid in phagocytosis. Experimentally, phagocytosis is very active for several days following radiation. A physician, writing in the Journal of Radiology in 1937 said that heat or poultices in no way could compare

with the effect of small doses of radiation in this type of condition. It is known that the more acute the inflammation and the smaller the dose of radiation, the better the result.

Many cancerous conditions of the dog are moderately radiosensitive and when the dog is admitted to our clinic, many of these lesions are quite large, but after two or three doses of intensive radiation, they become much smaller. The owner is quite sure that the treatment is doing good but actually little may have been done to the tumor, although the inflammatory reaction which accompanies a rapidly growing tumor has been decreased. This applies to any acute inflammatory lesion no matter where it is situated.

Although some uncertainty still surrounds the action of roentgen rays on inflammatory lesions, the weight of experimental and clinical evidence indicates that the first and major effect of the rays is the destruction of some of the infiltrating leucocytes, especially the lymphocytes. Destruction of the infiltrating leucocytes, especially the lymphocytes. Destruction of the infiltrating leucocytes tends to increase phagocytosis. In favor of this view is the fact that in the horse the rate of improvement, including relief from pain, which follows the irradiation of acute inflammatory lesions, agrees closely with the rate of leucocytic destruction induced in man and dog by exposure to roentgen rays.

In the case of an acutely injured horse, where an x-ray machine is not available, the usual procedure is to cool out the lesion and follow this with counter-irritation. This is a situation where cobalt or radium will give good results.

There are two forms of acute inflammation, one which follows the original injury, or the animal is hurt for the first time. The other follows perhaps a case of calcified tendinitis or a low, chronic bowed tendon which flares up after being re-injured — in other words an acute inflammation is superimposed upon a chronic condition. In these, there usually is a fair amount of calcium deposition. We tend to regard such injuries to the shoulder of man as calcified bursitis but in most cases there are cases of calcified peritendinitis or calcified tendinitis, where the supraspinatus tendon is attached to the humerus and tends to calcify because of injury.

Within the past few years, x-ray therapy has given good results in periarthritis and peritendinitis of the human shoulder. The presence or absence of calcification seems to be of some significance in determining the response to radiation. In subacute or chronic forms, the response is not so rapid and the end-result not as good as in the acute case. One of the striking effects of radiation therapy is the rather prompt absorption of calcareous deposits.

One worker, writing in Clinical Roentgen Therapy said: "Most clinicians now recognize that irradiation is the method of choice for bursitis and the end-result of roentgen therapy consists of substitution of an active circulation for passive congestion." This is getting back to Dr. Milne's remarks on hyperemia, induced by the injection of a chemical agent in contrast to the use of the firing iron. In the quotation above, however, the reference is to a calcified condition which is a chronic injury. "This reactivated circulation favors the absorption of calcium deposit".

Whether the calcium is resorbed or not, it is true in both human and animal patients that pain is often relieved. Many radiographs of human cases have been shown where after injury and treatment, the calcium formation is still present.

The older members of this organization will recall the calcareous lesion of tuberculosis in which there was a type of cheesy material, then a gritty type of substance followed by a hard calcified zone. Apparently the pressure and pain is caused by the cheesy and gritty, or salty material which is often reactivated when chronic injuries are re-injured. Radiation causes the resorption of this material.

To sum up:—

(1) The effect of irradiation in inflammatory conditions is due to the response to the destruction by x-rays of the leucocytes and lymphocytes, with the liberation of endotoxins which possess the power of phagocytosis and the production of hyperemia in the irradiated tissues. (2) It is known that normal circulation results in normal calcification. Increased circulation causes decalcification while decreased circulation causes an increase in calcification. The reason for calcification throughout the tendon attachments and in the bursa is the area of low metabolism because when circulation is decreased, increased calcification results. Anything which will bring more blood to the part will help to absorb the calcification.

MODERATOR JOHNSON: Dr. Chassels of Ontario is the next speaker and will consider the uses and mis-uses of radiation in horses.

DR. CHASSELS: My introduction to radiation therapy in horses was by a film and lecture by Dr. Leroy Johnson at the Stud Managers' course in Lexington, Kentucky. At that time radiation was administered by large x-ray therapy machines which were very expensive and not too practical for the average practitioner in the field. Dr. Johnson carried out extensive research in developing a small radium set which could be carried into the stall, and treatment could therefore be administered in a manner more suited to the needs of the practitioner in the field. Later, the application of radioactive cobalt was introduced.

I believe we all owe a great deal to Dr. Johnson for his extensive work in this field. He has tried to present his findings in an ethical manner, to responsible persons who have received training in the field. It is indeed frightening to imagine the possible damage that could result if this power fell into indiscriminate hands.

The Original Uses: Originally, radiation therapy was recommended for the treatment of: 1) inflamed tissues; 2) fibrositis; 3) calcification in its various forms; 4) arthritis; 5) exostosis; 6) tendinitis; 7) inflamed and torn ligaments; 8) carpitis; 9) splints; 10) buck-shins; 11) osselets; 12) ringbone; 13) navicular disease, by application of the navicular boot; 14) tumors; 15) cooling out of inflamed areas before firing.

Immediate exercise was recommended, along with therapy and actual racing was not considered harmful. In many cases, good results were obtained, some hopeless cripples being returned to racing form. Others showed no improvement.

My impression is that, when radiation therapy first became available to the practitioner, it was a fad. It was used on almost every imaginable pathological deformity. Because of its ability to dramatically relieve pain and inflammation in a short period of time, it was often used almost as a local anesthetic to relieve pain prior to a race.

Because it is available to licensed personnel only, it is my feeling that other practitioners resent this aspect, and probably recommend some type of therapy, such as the steroid group of drugs, and, if I may be so bold as to mention that frightening name, Butazolidin.

At the present time in Canada, and in other areas, it would appear that radiation therapy in equines is not being used very much.

Mis-uses: 1) It is my opinion that mild exercise is beneficial during the period of treatment, but actual racing is harmful. It just does not make sense to me to treat a leg undergoing inflammatory reaction and, at the same time, abuse the tissue by racing and fast work. We are all aware of the fact that time and nature play the greatest part in recovery, and all that the therapy is doing is assisting Mother Nature.

2) I do not believe that radiation therapy should be used as an argument against firing. I believe certain cases require firing and other cases indicate radiation.

3) Radiation therapy is not indicated as a quick solution to buck-shins. Certainly, it will relieve inflammation and soreness, in most cases in twelve to twenty-four hours, but I can assure you that most of these cases will recur.

4) In my opinion, radiation is not indicated in navicular disease and ringbone.

5) Radiation is not of great value in bowed tendons, except in very early cases, during the stage of infiltration, before fibrositis of the tendon sheath has occurred.

6) It is of very little value in suspensory ligament involvement.

7) For ethical reasons, it should not be used simply to remove soreness, to allow a horse to be used in a particular race.

Uses: 1) It is very useful for diminishing the inflammatory reaction prior to firing.

2) The best results I have had in radiation treatment is in cases of osselets. I am referring to cases in which the fetlock is swollen and, to the naked eye, it would appear that osselets have been formed but after x-rays have been taken there is no bone involvement. The diagnosis is fibrositis. Radiation will permanently cure most of these cases.

3) Radiation will also help dissolve certain small calcium deposits in joint capsules, especially the small floating deposits. I have followed some of these cases by a series of radiographs and have observed a reduction in size and, in some cases, a complete disappearance.

4) Sometimes the exostoses in cases of splints can be completely removed by radiation therapy.

5) Because of the complexity of the joint surfaces within the carpus of the equine, radiation therapy has been found to be particularly useful for the treatment of pathological processes.

6) I believe wonderful benefits are obtained by the radiation of joints following surgical removal of sequestra to arrest periosteum damage that is unavoidable in such surgery.

7) The benefits of radiation are well recognized in the treatment of cancer but I do not believe that this is too important in equine practice.

8) In certain cases of early bowed tendon and suspensory ligament strain, some benefit can be obtained by reducing the swelling and relieving pain, but the ultimate result is doubtful.

Conclusions: Radiation therapy has been used, proved and recognized in the human field. To me, it is a pity that veterinarians do not recognize this and utilize this product in the proper manner. It has gone through the fad stage where it was overdone. Now it is not being used enough. I would like to see this therapy fall into its proper category and the wonderful advantages it has to offer, utilized.

Perhaps we should review our system of availability, bearing in mind however, that the handling of such a dangerous product must be guarded. Perhaps we should review the mosts of therapy. Perhaps we should organize or reorganize quite a few aspects of this therapy, but let us return to using it and have our animals benefit from the useful properties it has to offer.

MODERATOR JOHNSON: The next speaker is Dr. Proctor who will discuss, he tells me, the historical aspects of radiation therapy.

DR. PROCTOR: My interest in radiation therapy began in 1947 when I went to the Miami meeting of the American Veterinary Medical Association, and heard Dr. Thom give an address which described essentially what he said to us a moment ago.

My first personal experience with radiation therapy was when I learned, that by radium application, the son of a prominent trainer had been relieved of painful shin splints incurred while playing basketball. So I thought, "This is great; I will try it." I borrowed a plaque of radium from a dermatologist friend and, following a hit-or-miss system, I attempted to radiate a splint. I did a whing-ding of a job! I got an area just as big as my plaque! First of all there was vesicle formation and swelling followed by an ulcer, and it took six months for this area to heal. My intitial attempt at radiation therapy was a dismal failure, and I became very conservative on the subject. I helped the splint, but there was vesicle formation, necrosis, and the ulcer. Finally, when the burn healed, there remained a large flock of white hair on a nice show horse!

At the Miami meeting, Dr. Thom gave a very complete paper, with several case reports, on the use of x-rays in relieving inflammatory processes in the horse. The clinical results were confirmed by Meginnis at a later date, but it was so impractical and potentially dangerous to those in the vicinity that Drs. Johnson and Henschke devised a method of using radium sticks for gamma radiation therapy. This meant we could ambulate with our radium. Also, the radium was applied in such a way that we got away from the undesirable effects of alpha and beta radiation. These are the so-called soft rays that cause superficial tissue damage and discoloration, or the growth of hair without pigment.

I believe Dr. Howard White and I leased one of the first sets of radium used in practice. This was in the winter of 1954, if I am not mistaken.

Radium is a powder and, due to the danger of accidental contamination if a capsule should be broken, it was used with some misgiving. In other words, if one broke a capsule of radium, the radium powder was scattered. There is a lovely four-story building in Cincinnati right now that has a big padlock on the door. No-

body can go in because it is completely contaminated with radium powder. This building is an economic liability and will continue to be for some time because it has been padlocked. This is the type of potential danger involved in using the powdered radium.

Radioactive cobalt became available in 1956. Physically, the radioactive Cobalt, so-called Co^{60}, was in the form of small cylinders. These small cylinders resemble the flint in a cigarette lighter.

Drs. Johnson and Henschke prepared six elongated sticks, each containing six pellets of radioactive Co^{60}. They were arranged in such a fashion that, depending on the strength of the set, 125 to 150 rems were liberated over an 8-hour, 12-hour and, finally, a 24-hour period. The length of time that these r's or rems were disseminated was based on the power of the set, and it had to be cut down progressively because of the increasingly stringent regulations of the Atomic Energy Commission. We might break a stick and lose the radioactive cobalt pellets but no longer were we faced with the problem of contamination from radioactive powder as formerly. Also, the fission product, Co^{60}, was not as expensive as radium itself. However, to me as a practitioner, a much more annoying problem developed. Since radioactive Co^{60} is an atomic fission by-product, one must procure a license to obtain and use it. The Atomic Energy Commission requires a licensee to abide by a 30-page code, and this is written in true bureaucratic jargon that even a Pentagon lawyer would have trouble translating! Failure to comply with this regulation is a federal offense, with a large fine and incarceration, under federal supervision, promised in no uncertain terms!

Radioactive material in storage vaults must be clearly marked with signs of uniform dimensions as set forth by the Atomic Energy Commission.

High radiation areas are defined, and must be placarded to comply with the regulations. In other words, if you are using it in a horse's stall, you have to say, "Here is radioactive material. Here is a high radiation area."

All personnel must wear monitoring devices, and levels of radiation are computed while the cobalt is in storage and also in use. In other words, we had to have a monitoring device on each man to show approximately how many millirems he picked up during a 30-day period. This was a matter of permanent record.

The storage vault was a solid concrete block in the ground, with a 4-inch concrete lid on it, which took four men and a boy to lift. The cobalt was in a lead pig that weighed 150 pounds, and this was dropped down into the vault. It was placarded. I had to get a certified reading by a prominent physicist that the millirems at the level of the container were so much; the millirems at 10

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feet were so much. We built a fence around, and put a padlock on it.

Then we did the same thing with the material we placed on the horse. Bear in mind, this is not kid stuff; this is a federal regulation. It is backed up by a $10,000 fine and six months in jail.

Records were required on all movement of cobalt. Any loss or accident involving the material must be reported to the Atomic Energy Commission. Correspondence covered reams of paper.

All this might have been acceptable except that I am an equine practitioner. I float teeth, practice medicine and deliver foals. I treat sterile mares, sick foals, and try to do veterinary surgery, as well as radiating chronic inflammation.

In short, gentlemen, the Atomic Energy Commission was regulating me out of business, but by this time, the use of cobalt as part of my post-operative orthopedic procedure had shown enough promise that I felt that losing my Atomic Energy Commission license would jeopardize the success of the surgery.

I feel that the inflammatory process, followed by calcification is a species reaction whose intensity is peculiar to the horse. I do not know of any other animal, or man, in which there is this characteristic calcium deposition. The phenomenon can be arrested and, to some extent, reversed by gamma radiation.

I have had ankles that I wanted to fire from September to December, and 500 or 1000 r will get those ankles ice cold, and I can go ahead with the firing procedure.

Gamma radiation fills a gap in this type of therapeutic procedure where there is no other answer. The use of cobalt in cooling out joints is dramatic. Successful use is dependent on knowledge of the etiology of the condition and removal of the cause.

I agree with Dr. Chassels that you cannot treat buck-shins and break the horse out of the gate next morning and expect any good results.

Radiation will not cure a condition when the etiology is present and being maintained every day. This is just common sense. Rest is a necessary part of the procedure, if lasting remission of symptoms is desired.

I like to use cobalt therapy in splints. I like to use it in buck-shins. As a permanent cure, radiation therapy plus rest has proved to be well worthwhile.

I have arbitrarily decided on a dosage of 1000 r in a six-week period. Dr. Thom, in 1946 or 1947, recommended, I think, as much as 1500. Being essentially a conservative type of person with something I didn't know much about, I said if 1500 would

do good for him, maybe 1000 would work for me. That has been my procedure for fifteen years, and I have got results and am not going to change.

I give 1000 r in a six-week period. I may use it six days straight and wait six weeks, use it three days on and three days off, or give it daily, once on every Monday, for six consecutive weeks. I am not as dogmatic about my results as I might be, but I will tell you this: I have never damaged a horse, and the only time I got discoloration or vesicle formation was when an accident happened, and the end of the cobalt cylinder was actually in contact with the skin and hair.

You can use it at this dosage rate and I will guarantee that you will never do any harm. One thing that can be said for Co^{60} is that it has an excellent quality of gamma radiation. It is a hard ray. They tell me, and this is hearsay, that as far as penetration is concerned, the gamma radiation of Co^{60} is equivalent to a 1,000,000-volt x-ray machine.

Since the source of radiation is portable, and much more powerful, as far as genuine gamma radiation is concerned, than the most powerful x-ray machine that any ordinary practitioner could possibly have access to, the advantages and practicability are obvious. In addition, there is no alpha or beta radiation to worry about, therefore, the soft rays which cause skin and hair discoloration are at a minimum.

I am very happy to be able to put down in black and white Dr. Thom's opinion of how radiation exerts its effects, but I did take exception to one statement. He said he felt there was prompt absorption. It may be that there is prompt absorption of the caseous material but I cannot say that there is anything dramatic or prompt about the reaction that I get with radiation therapy. I like to tell my clients—and this, again, is supposition on my part—that if it took six months for the lesion to get there, it is going to take six months for it to go away. Another thing; visualize an icicle that is sharp and jagged; if you hold it in your hands for five minutes, you no longer have something that is sharp or jagged but you are going to have a lump of ice.

I feel that rest causes some resorption of the sharp spicules but the major portion of the calcium is going to remain.

I also want to agree with the statement that increased circulation causes some degree of calcium absorption. We had a lovely gray horse which had a basal fracture of both sesamoids. There was no chance that surgery would help, so rather than just give up on him, we used a series of the most severe blisters that I have ever put on a horse, over a period of 120 days, and I have

the x-rays which won't leave any doubt in anybody's mind but that prolonged, severe, deep, combination blister and sweat applications of an iodine absorbent will definitely cause some resorption of calcium.

I told you about the dramatic cessation of the cardinal symptoms of inflammation. You can take an ankle that has been hot for ninety days and have it cold within forty-eight hours. This is an outstanding characteristic that is noticed with the use of cobalt.

Here, again, I will agree that sometimes, even though on x-ray examination you do not see any difference in the overall picture, suddenly, instead of having a chronically lame horse with acute inflammation, the joint is cool and the horse sound and the chances are he will stay sound. I believe you can gallop him or go into slow work indefinitely. If you keep him away from the race track, you will have a sound horse from then on. You cannot expect radiation therapy to keep a horse sound and keep him in training at the same time. Nothing takes the place of rest and Mother Nature.

MODERATOR JOHNSON: Our next speaker is Dr. C. F. Reid, who, as an A.V.M.A. Research Fellow, has studied the physical and technical aspects of radiotherapy. It was felt that his contribution to this panel would be a valuable one.

DR. REID: For the past two years I have been at the Veterinary College of Cornell University, engaged in a research program involving radiation therapy in the horse. The material I will present is part of this work.

In the time alloted, I shall deal with two related subjects: (1) Considerations of treatment planning when radiation therapy is to be used as a modality of treatment, and (2) protection and the responsibility of the operator who uses sources of ionizing radiation.

All of you, I am sure, have encountered clinical cases at one time or another in which you probably wondered if radiation therapy could be of benefit to the animal. Various reasons, including the unavailability of sources of ionizing radiation, inexperience in the use of ionizing radiation and word of mouth talk of unpredictable results when ionizing radiation is used, have prompted you to employ more conventional methods of treatment. Accordingly, I will discuss what is to be considered when one contemplates the use of ionizing radiation as a modality of treatment.

The use of radiation therapy is dependent on three major consideration: (1) diagnosis, (2) dose to be delivered, and (3) method of delivering the radiation.

The most important single factor related to success in the treatment of a condition is proper diagnosis. This is true no matter what modality of treatment you intend to pursue. The success or failure of radiation therapy is dependent on proper pretherapy diagnosis, because the condition to be treated has to be amenable to radiation therapy.

How does one know if a condition will respond successfully to a course of radiation therapy? There are two sources to which we can refer when deciding if the condition can be successfully treated by a series of radiation treatments:

One, of course, is past experience in human medicine. We should recall that the initiation of the use of ionizing radiation in veterinary medicine, particularly in equine lameness, was due to the fact that a large portion of human radiotherapy deals with the treatment of such conditions as bursitis, peritendinitis, arthritis, and tendosynovitis — all of which are at the top of the list of equine problems. It is reasonable to assume that similar conditions in horses would respond to such treatment. We therefore rely on previous human experience when deciding whether radiation will be of help in treatment.

Drs. Johnson, Thom, Reed and others have had extensive experience in treating many varied conditions and have knowledge with regard to those which respond and those which do not, so we can now rely on previous experience in the treatment of certain equine lamenesses.

In addition to those two sources of information, radiation therapy has also been shown to be of great value in the prevention of post-operative complications following bone surgery.

Once the condition is diagnosed and radiation therapy is to be used, the proper dose has to be determined. This is done by reference to the results of human and veterinary research. Since many parallel conditions in the human have been treated for many years with good results, we can use such treatment regimes as a baseline for our treatments. We can also draw on our previous knowledge in the actual treatment of horses.

With regard to the measurement of amounts of ionizing radiation, and since time does not allow an extensive dissertation on nomenclatures of amounts, I will use the term "r" (roentgen) to signify amount. I realize there are other terms such as rad, etc. but defer from including them in this discussion.

There are certain limitations with regard to the dose. We do not want to give such a high dose of radiation that the horse ends up with necrosis of the skin and other sequellae of radiation

treatment. In human medicine the amount of ionizing radiation that the skin can tolerate is well known. This, however, is not true in veterinary medicine. A radio-therapist in veterinary medicine does not know the maximum amount of radiation he can deliver to the skin of a horse and yet not encounter difficulty.

Consequently, it is necessary to know if the calculated amount will be tolerated by the animal without secondary radiation damage. It is essential to be aware of how much of the various kinds of radiation a horse can withstand and what changes will be encountered if these perimeters are exceeded. In order to determine these skin tolerances in horses, certain experimental exposures were carried out.

Investigation was carried out on the epilating dose, de-pigmentation dose and necrosis dose. A total of six animals were irradiated in fifty-one areas. All these animals had approximately the same skin tolerances. (Figs. 1, 2, 3).

To insure accurate dosage, the animal was restrained on an operating table while under treatment. Non-fractionated doses of x-ray irradiation (280 KV, HVL 1.5mm of copper) were administered in the following doses: 2000r, 2500r, 3000r, each in a 10cm by 10cm field — all at the same sitting.

The following conclusions were drawn: (1) If a dose of 1000r or less of x-ray irradiation of 280 KV is given in one non-fractionated dose, no gross disturbance of the hair coat or skin will be noted. (2) a dose greater than 1000r, but not exceeding 2000r will cause hair change from pigmented to non-pigmented hair, and (3) if 2000r non-fractionated dose is exceeded, there is a likelihood of skin necrosis.

These experimental facts enable us to have a baseline for treatment planning. In other words, we know the maximum amount of radiation the skin of a horse can tolerate. It is necessary to stress that this applies to a single (non-fractionated) dose at 280 KV (HVL 1.5mm of Cu). What happens if we split this 1000r into three equal doses? How much radiation can we deliver to obtain the same results as with 1000r in a single dose? In other words, what happens if one fractionates this dose? Fig. 4 applies to human skin but the findings have been found true in animals as well. With this information we can more efficiently determine the necessary, yet safe dose.

What happens if we use radium or cobalt to deliver this dose? The gamma ray energy from radium and cobalt is about 1200 KV in comparison to the 280 KV at which level this work was done. What happens to the skin tolerance now? When higher energy radiation is used, skin tolerance increases. In other words,

it takes more radiation to cause the same effect. For example, according to human research if at 280 KV (medium energy radiation) you give 2000r, you would have to give 25% more or 2500r to achieve the same effect when using high energy radiation.

With this further information one can determine better whether the animal's tolerance to radiation will be exceeded with resulting secondary radiation sequellae as demonstrated in Fig. 1--3. The effect of pigmented and non-pigmented skin is important. Exposures of irradiated animals to sunlight enhances the effect of irradiation. All these various perimeters have to be considered when one is going to irradiate an animal.

Since we know now the diagnosis and the amount of radiation to treat the condition safely, the next problem is how to deliver ionizing radiation to the affected area.

For a clinician to successfully predict the results of radiation therapy, he must, as mentioned earlier, diagnose the condition, decide on the dose to be delivered for an effect and deliver that dose to the part. If any one of these three is done incorrectly, one is unable to predict satisfactorily the end result. The problem now is to deliver a dose of radiation which will be of value. I feel that the unpredictability of results when using radiation therapy is due to the inability to predict the dose which is delivered to an area. If a specific lesion needs 1000r and receives only 500r, one can appreciate how uncertain the results will be.

Most of the radiation used in treating such conditions in the human as bursitis, arthritis, and tendosynovitis, etc., is delivered by using 140 - 250 KV x-ray. The patient lies on a table in an appropriate position and the bulky x-ray tube is placed in position and the treatment is completed after all personnel leave the treatment area. For this type of therapy, large expensive bulky equipment is necessary. A separate lead-shielded therapy room is required, and the patient has to remain perfectly still during the exposure. If the area being treated moves much during the two to ten minute treatment period, the dose to the area is altered. This variation in the dose can be quite significant and can affect the outcome of the treatment.

In the treatment of horses, some veterinarians use this expensive and bulky x-ray therapy equipment. Animals to be treated are placed in front of the x-ray machine and the tube is positioned over the affected area. When treating thoroughbreds, it is quite difficult to get them to stand perfectly still in one position for any prolonged period of time. For this reason there is some movement of the animal's injured leg during exposure or the leg was not placed at the proper distance from the tube before treatment began. What is the significance of this movement?

Radiation falls off inversely as the square of the distance. This means that if a horse's leg should be 10cm from the focal spot of an x-ray tube when the dose delivered is 100r per minute, but is actually at a point 20cm from the tube the delivered dose will be only 25r per minute. This can quite significantly affect the results of the treatment because the dose actually delivered to the lesion is nowhere near the intended dose.

The difference between air dose, surface dose, skin dose and back scatter also merits consideration. Fig. 5 illustrates these four points. Air dose is self-explanatory, skin dose and surface dose are synoymous terms and back scatter is the percentage increase of air dose that gives the surface dose due to secondary radiation of the tissue being irradiated. Therefore, in conventional x-ray therapy, surface dose will always be higher than air dose. If these factors are not considered, one is not able to predict accurately the dose of radiation being delivered to an area.

To own and operate an x-ray therapy machine for horses is quite expensive and cumbersome. The unit is completely immobile. The animals have to be brought to the therapy area and it is difficult to position them for correct treatment. For this reason, other more mobile sources of radiation were sought so that radiation could be delivered more easily to an area. Dr. Henschke (M.D.) a radiologist and Dr. Johnson at Ohio pioneered in the use of radium and cobalt as sources of ionizing radiation for the treatment of equine lameness. Radium and cobalt, which are unstable radioactive substances, try to reach a stable form by throwing off gamma rays. These rays are the same as x-rays as far as the effect on tissue is concerned. In these elements we have mobile sources of radiation for use in treatment. Let us remember that the radiation from these sources is with us all the time and cannot be shut off like the x-rays from a machine. A potential hazard exists all the time. One must be able to predict the dose being delivered to an area and this can be a major problem.

The radio-active material is placed into applicator tubes which are then placed about the area to be treated. The positioning of these applicators very markedly affects the dose that the area will receive. In other words, if the applicators are grouped together around the medial splint, the splint will receive "X" dose, but if they are spread out equally around the leg, the dose to the medial splint might be X/2. Since protective covering varies among animals, a radiation boot was designed with actually holds the applicators. This boot has a fixed circumference and diameter and is available in various sizes. The position of the applicators can be changed and reproduced repeatedly (Fig. 6). These radiation boots are calibrated with a victorein r chamber on an artifical or phantom leg. Any geometrical arrangement of the applicators in the boot can be made and the dose determined in the

artifical leg. Therefore, after these boots are calibrated, one can predict the dose delivered to a part.

What kind of exposure does the clinician receive when using these radio-isotopes and how can he protect himself?

In protecting oneself from ionizing radiation, three important principles are followed: (1) distance, (2) time and (3) barriers. How these three factors are utilized depends upon the energy and intensity of radiation encountered. First: When I was speaking of x-ray therapy in the horse, I mentioned that radiation falls off inversely as the square of the distance. The farther one can stay away from a source of radiation, the lower will be the exposure. Therefore, in using these isotopes, one never handles the applicator tubes with the hands but always uses forceps at arms' length. The next consideration is time: the less time spent near sources of radiation, the less exposure will be received. The boot was designed so that an individual could use it at a distance and with speed. The average loading takes less than two minutes and unloading is even more rapid.

The third protective device is a barrier. Barriers can be made of lead, steel, concrete, etc. We use lead a great deal because of its heavy density and its ability to stop radiation photons. Lead barriers are very effective against low to medium energy x-rays (50 KV - 250 KV), and the lead barrier can be relatively light, but when lead barriers are used for protection from high energy photons from radium and cobalt the thickness of the barrier becomes tremendous. For instance, if a source of radium (1200 KV) emits at point A, 100r per minute and one wants to put a lead shield in front of the source so point A will read only 50r per minute, a lead shield from 12 - 15 mm thick would be required to cut the radiation down by one half. The average lead gloves used for x-ray protection are 0.5mm thick which means the dose to the hands is reduced by practically nothing (3%). However, gloves are cumbersome and the time spent in handling the applicators is markedly increased thus the operator is exposed to a higher dose of radiation. Lead barriers, however, are important in the storage and carrying of applicators to the stalls where the animals will be treated. The storage container is 3 inches thick and the carrier 1⅝ inches thick. This considerably cuts down the dose, but there is significant radiation coming through the container so exposure is evident even when the applicators are in the containers. You might wonder what exposures an operator receives while putting on and taking off the applicators. The total body radiation from the complete operation of putting the boot on, taking it off and carrying the radioactive applicators to the stall is less than 6 mr. The actual exposure to the hands, while using them, is less than 10 mr, which means that an operator can do 10 complete operations per week and still be within A.E.C. regula-

tions. A.E.C. recommendations are that radiological workers do not exceed 100 mr per week total body exposure and 1500mr per week to the hands. Non-radiological workers such as grooms, trainers, secretaries, etc. can receive only 10% of this dose or 10 mr per week total body exposure and 150 mr per week to the hands. It is the responsibility of the clinician using radio-therapy to see that innocent by-standers do not get excessive radiation exposure. This becomes a problem when using x-ray therapy for someone must hold the horse while it is being irradiated. Proper barriers should be used.

It is also the responsibility of the operator to maintain permanent records of exposure of personnel involved in radiation therapy work. This can most easily be accomplished by the use of film badges which give a permanent record of the total body exposure. Also, such things as dosimeters can be used. They give an immediate reading of the amount of exposure during a single operation and an accurate check can be made by employing both devices.

DR. TEIGLAND: Some years ago I used radium and also cobalt. When treating a case, one day, I noticed that there were only five sticks in the applicator instead of six. Somehow, one of them had disappeared. Apparently, the horse had taken hold of the tip of one of the sticks. We searched for three hours without success. I called the radiologist from Jackson Memorial Hospital and he came out with a very fancy instrument which I believe is called a "Q.T.Pi." Six hours later, after covering seventeen acres of manure at the back of Gulfstream Park, we found it four feet beneath the surface.

Fires have been known to occur at race tracks and I wonder if the sticks would remain in place if pandemonium broke out and the horses bolted. I think this merits consideration.

DR. CHASSELS: I was quite surprised to hear of Dr. Proctor's difficulties with the Atomic Energy Commission. I have not experienced anything like that in Canada. I am assuming that the same difficulties would exist while working at the track. What is your opinion of this and what is the ultimate outcome of cobalt therapy at the track?

DR. PROCTOR: Horses to be treated are put into a solid concrete block stall, which is more or less isolated. The liability rests with the operator and I do not see how one can ambulate with this material, have these horses under outside supervision and not get into trouble eventually. I think that those who have used this material will bear me out when I say that everything conceivable can happen — not only can happen, but does. I use the material on my farm, on my own premises and nowhere else.

DR. C. F. REID: To use this material, one has to be licensed by the A.E.C. In other words this body regulates Cobalt 60 in regard to how it will be used in treating horses. The A.E.C. is not responsible for regulating the use of radium and its derivatives. If radium is used for therapy, the regulations for its use comes under the jurisdiction of the individual States in which it will be used and therefore the controlling body is the State Department of Health.

DR. PROCTOR: I found it quite impracticable to employ this form of treatment in the usual horse stall. For this reason, I gave up trying to ambulate with it, and prepared a concrete stall where it could be used in compliance with the regulations. It is a valuable asset to me in post-surgical treatment.

MODERATOR JOHNSON: I was just going to point out to Dr. Proctor that radium does not come under control of the A.E.C., so it can be used but there are potential dangers. I think it should also be pointed out that a chart accompanies the sets that are available, so there is no figuring of a dose. The leg is measured in centimeters and from that one knows the skin dose, the average tissue dose and the dose on the surface of the bone.

QUESTION: I would like to ask the cost of one of these cobalt units.

MODERATOR JOHNSON: The name of the company and the individual who manufactures these units is Technical Specialties Incorporated, 4608, High Street, Columbus 14, Ohio — Mr. Pierce. I can't tell you what the charges are but he can.

QUESTION: I would like to ask Dr. Reid if he feels that the so-called ambulatory units have any distinct advantage over the heavy, fixed x-ray units, keeping in mind the lesion to be treated.

DR. REID: The energy of the radiation used is very important. Actually, I do not have any real experimental evidence, but just from my own experience, the actual high energy of cobalt and radium I think is too high. You would be much better off using medium energy x-ray and delivering more superficial doses to the superficial tissues that are affected, but this is impractical due to the fact that these x-ray machines are very expensive and horses have to be brought to them for treatment. Since we are trying to treat fairly superficial structures, the lower the energy of the radiation, the more ionization occurs in the superficial structures with a resultant beneficial effect.

QUESTION: I had a long conversation with a gentleman from Michael Reese Hospital who some years ago had radiated some neuromas that had been induced in saddle horses. In his opinion, the veterinarian was on safer ground using the heavier, in-place

units. He said that he had had one bitter, frightening experience that kept him awake all night worring about it. What is your feeling about this?

DR. REID: You have to be cognizant of the dangers involved in the ambulatory models. You also have to be cognizant of the exposures that individuals are receiving, when they use ambulatory radiation devices. In regard to x-ray therapy, individuals holding the animal being treated have to be protected by adequate lead barriers or they will be receiving excessive radiation.

DR. THOM: May I answer part of that? Up until about ten years ago, we used radium exclusively. Since that time we have used 220 kv. x-ray. The regulations in California are relatively strict. The control panel can be arranged so that the handler can be protected, within acceptable limits, from scattered radiation. We run constant monitoring checks to ensure this. The horse may move somewhat but our dose now is around 1200 r, total dose delivered. In this area, if there is some back-and-forth movement, the amount is still within the epilation dose and we rarely get epilation.

We have to remember also that the other leg is likewise receiving some radiation. If you intend to treat this other leg as well, allowance must be made for the radiation it has already received. Panels can be arranged very simply so that the person holding the horse can stand and peer through a lead glass window. After the horse has been treated for the first time, there is very little trouble.

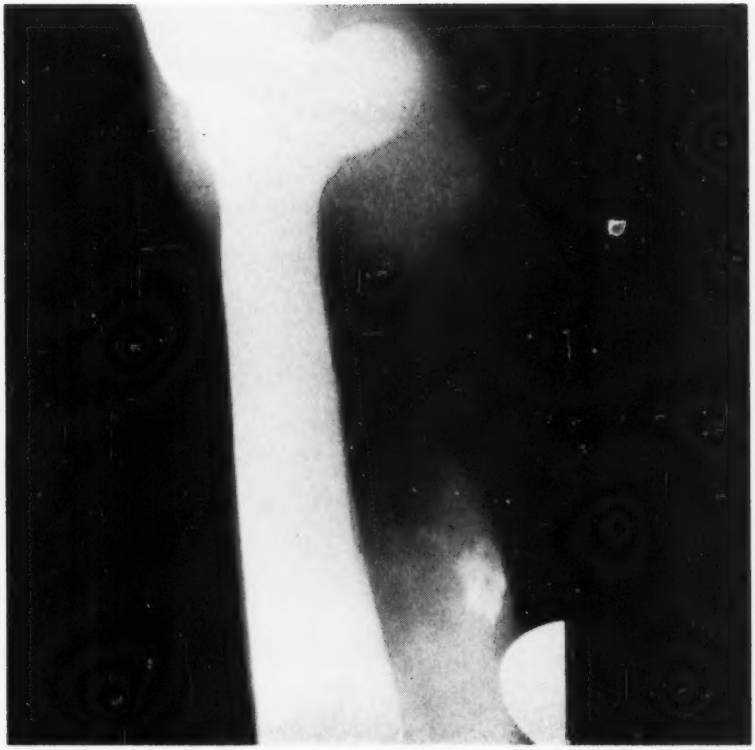
We used radium in quite a different manner. We used it in a little bomb which we had to move around. Although I believe that the sticks are certainly better, we felt that there was too much exposure of the fingers to the radium and that if the x-ray unit is properly set up, it is far safer than the ambulatory radio-active elements can ever be, because of the difference in the voltage. We can control what we are doing.

MODERATOR JOHNSON: Are there any more questions?

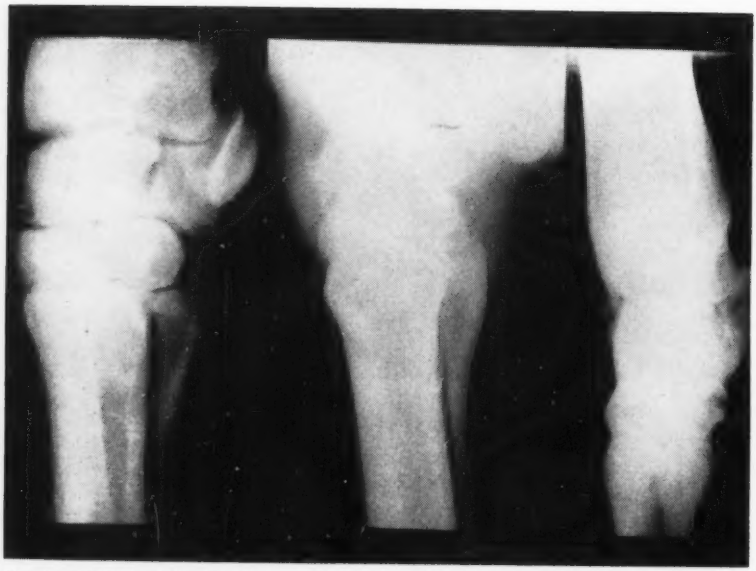
DR. CHASSELS: Dr. Johnson, is there anything we can do to reduce the cost of this type of therapy? I think cost may be one of the reasons why it is not being used as much as it should be.

MODERATOR JOHNSON: That is strictly up to the manufacturer. Originally, he was under a great deal of expense, because most of the units were custom made. He had to experiment so somebody had to absorb the cost.

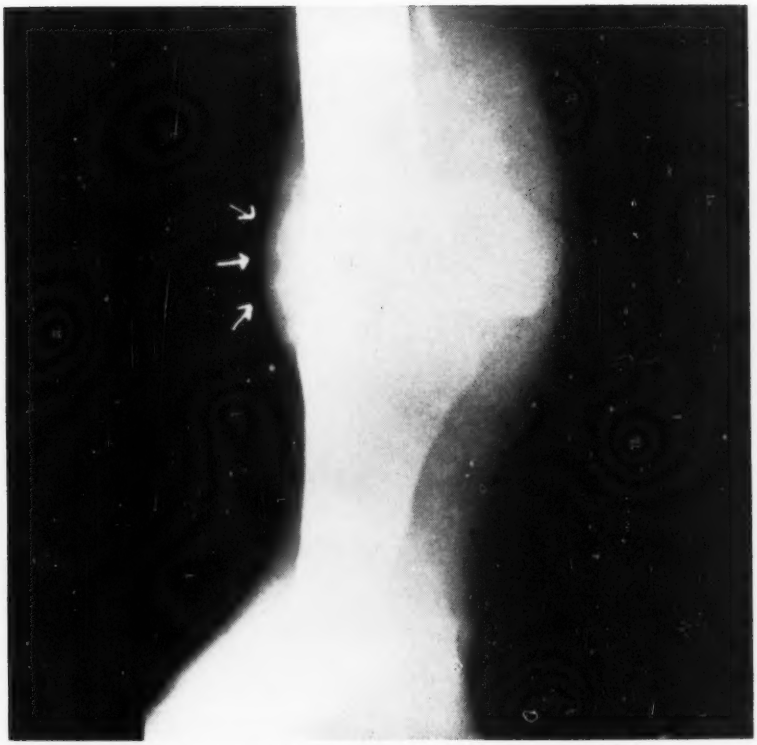
If there are no more questions, we will conclude this part of the program.



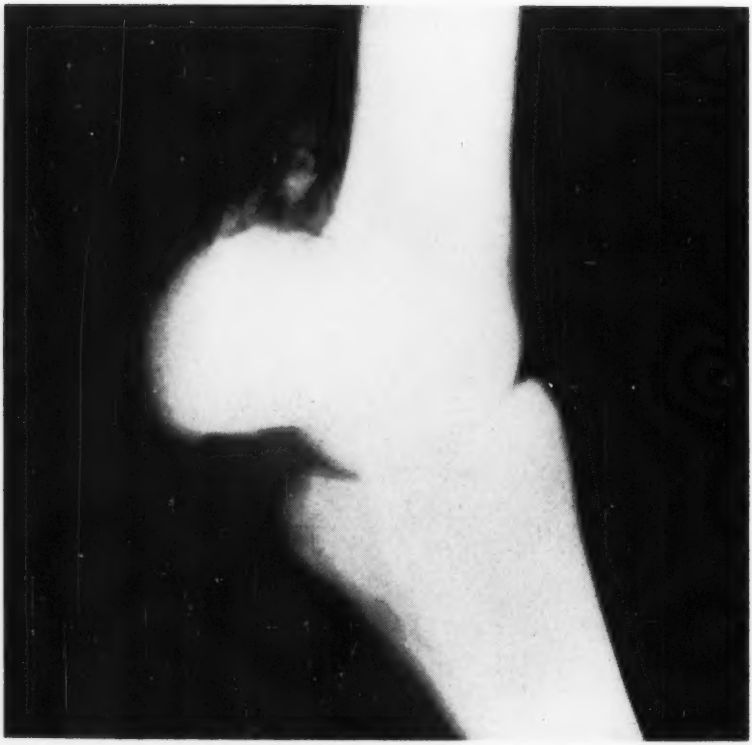
M. L. THOM. Fig. 1. Calcified hematoma in a long-standing, bowed tendon.



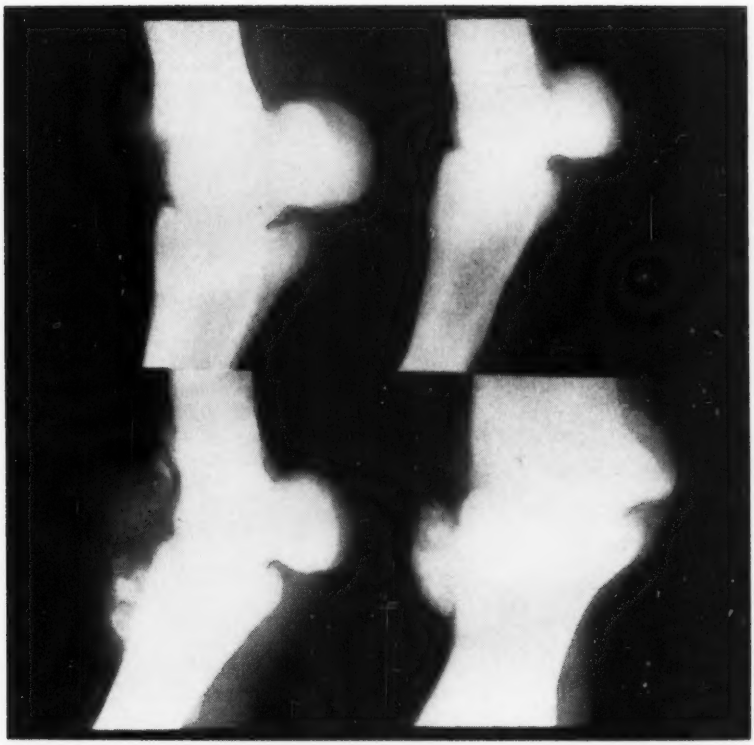
M. L. THOM. Fig. 2. Carpal lesions which may be favorably influenced by radiation.



M. L. THOM. Fig. 3. Typical calcification within the fetlock joint capsule. At this stage, pain will be relieved by radiation therapy even if the calcium is not absorbed.

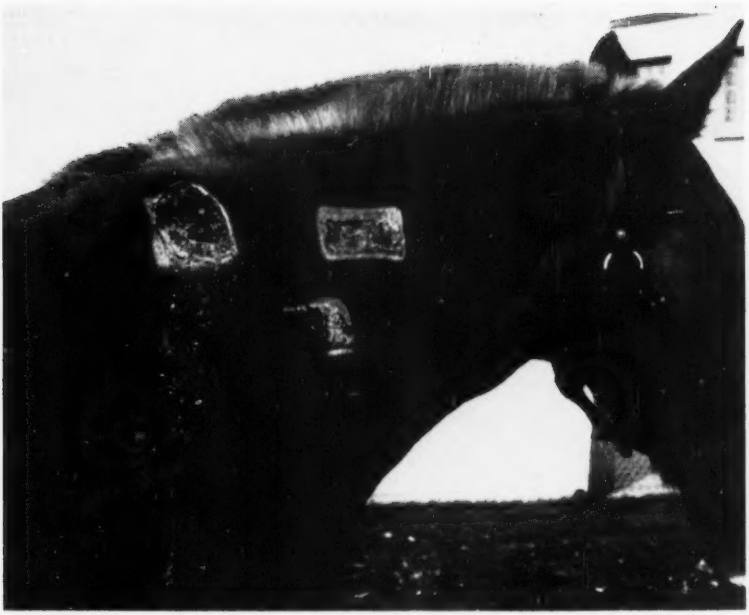


M. L. THOM. Fig. 4. Calcification within the suspensory ligament. If this case had been irridiated after injury, deposition of calcium would have been unlikely.

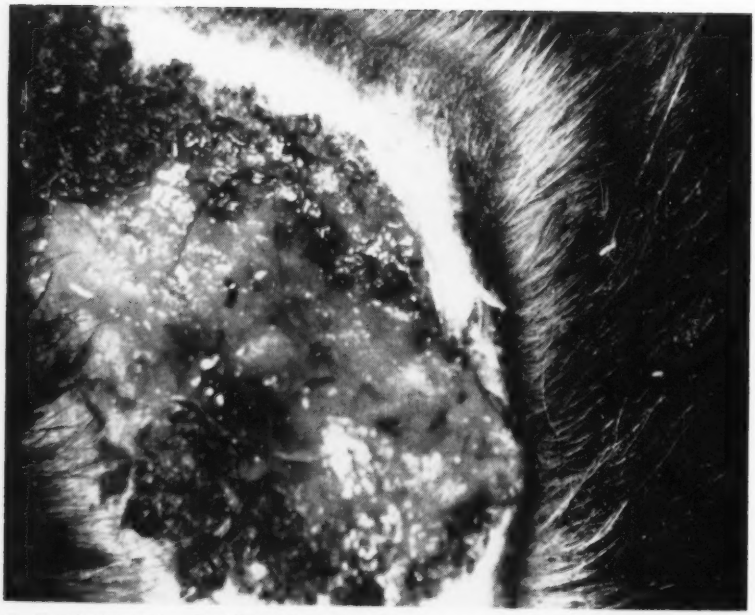


M. L. THOM. Fig. 5. Stages in the progression of calcium deposition following injury to the region of the fetlock. The two top cases are likely candidates for radiation therapy.

C. F. REID. Fig. 1. Sixteen days after radiation. Note the edematous plaques; the skin actually becomes wrinkled.



C. F. REID. Fig. 2. One hundred days after radiation. Severe damage at 3000r but hardly any at 2500r.



C. F. REID. Fig. 3. Necrosis at 3000r, 215 days after radiation. Healing will take about one year.

XUM

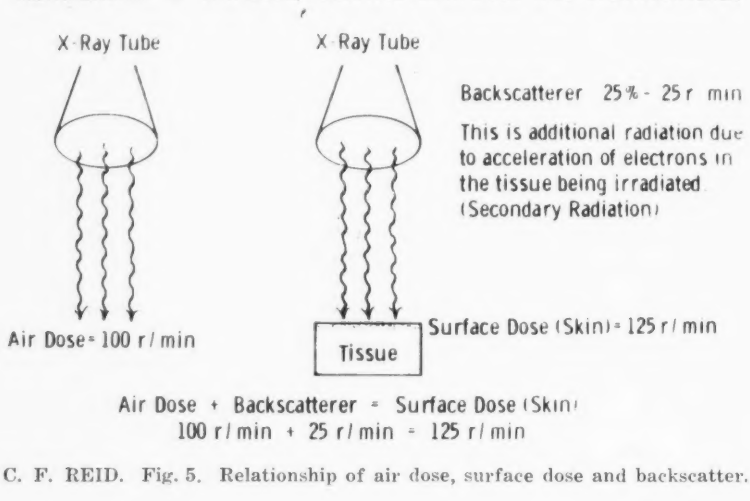
VARIOUS FRACTIONAL DOSES WHICH PRODUCE THE SAME EFFECT IN HUMAN SKIN

(Reisner, A.: Ergeln. d. med. Strahlenforsch, 6: 1933)

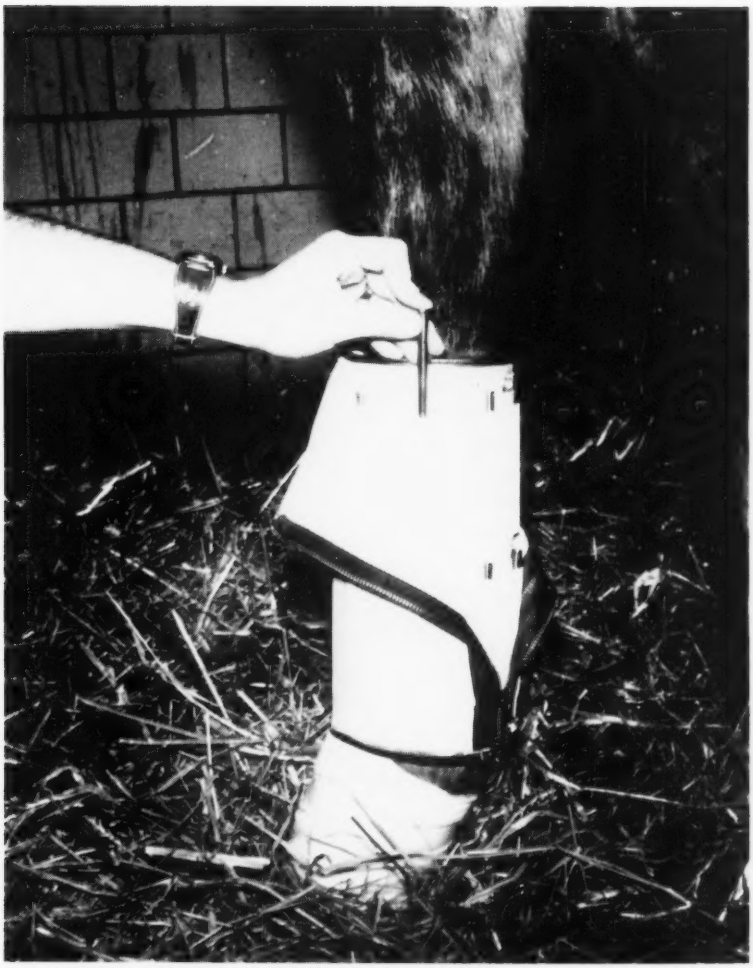
No. of Irradiations	No. of Roentgens per Irradiation	Total Roentgens	Per cent of Total Dose
1	1000	1000	100
2	650	1300	130
3	500	1500	150
4	400	1600	160
7	300	2100	210
12	200	2400	240
27	100	2700	270

C. F. REID. Fig. 4. "If 1000r are given at one dose, erythema results. When 500r are given daily for three days, a total of 1500r is necessary to achieve the same end-point as is caused by a single dose of 1000r."

Acknowledgement is made to the publishers of Radiology for permission to reproduce this chart which is taken from an article "The Rate of Recovery of Human Skin from the effects of Hard or Soft Roentgen Rays or Gamma Rays" by W. S. MacComb and E. H. Quimby in Radiology 27(1936) p 196.


RELATIONSHIP OF AIR DOSE, SURFACE (Skin) DOSE AND BACKSCATTERER
X-Ray Tube
X-Ray Tube
Backscatterer 25% - 25 r min
This is additional radiation due to acceleration of electrons in the tissue being irradiated (Secondary Radiation)
Air Dose = 100 r/min
Tissue
Surface Dose (Skin) = 125 r/min
Air Dose + Backscatterer = Surface Dose (Skin)
100 r/min + 25 r/min = 125 r/min

C. F. REID. Fig. 5. Relationship of air dose, surface dose and backscatter.



C. F. REID. Fig. 6. Applicators being placed in radiation boot.

FLUID THERAPY IN THE HORSE

J. E. Burch

There is a universal awareness of the need to recognize and correct imbalances of fluid and electrolytes in many metabolic and infectious diseases, and also stress conditions of horses. Dr. Charles Bild has stated that a starving animal can lose nearly all of its glycogen and fat, half of its body protein, and two-fifths of its body weight, without death occurring, but the loss of ten per cent of the body water results in a serious metabolic disturbance and a loss of twenty-five per cent almost inevitably means death.

A thorough understanding of fluid therapy is not casually acquired, and the ramifications are complex. Most equine practitioners see each day horses that live in a state of water, electrolyte, and/or protein depletion.

Water deficiency is most commonly encountered when attendants fail to give horses enough to drink, or there is excess loss through the gastrointestinal tract, kidneys, lungs, or skin. In a case of simple water loss, when enough water is not consumed, the extracellular fluid becomes hypertonic. There is a subsequent shift of water from the cells by osmosis.

Electrolyte deficiency arises when the intake is inadequate or metabolism is altered or excess loss occurs in disease and/or stress conditions. In electrolyte depletion, the extracellular fluid becomes hypotonic and there is a subsequent shift of water into the cells and the extracellular volume falls. The kidneys continue to excrete water in an attempt to maintain extracellular isotonicity. Water excretion is under the hormone control of the posterior pitutary gland and the adrenal cortex. The latter controls electrolyte excretion.

Dehydration is the result of either water or electrolyte deficiency or both.

A most important practical point in the difference between the maintenance of water and electrolyte balance is that the water intake is controlled by thirst, but there is no similar warning of slight electrolyte deficiency, until the point is reached where the horse shows anorexia, muscular disturbance, reduced sweating, and lack of thirst. This problem is commonly associated with hypoproteinemia from inadequate intake, absorption and metabolism. In Florida we see horses that sweat very little and consume inadequate quantities of water even after vigorous exercise. Many

of these horses have sustained electrolyte loss over a long period of time.

It is important to obtain the history of the animal in order to ascertain the type and amount of fluid or electrolyte loss in the individual case. If a thousand pound horse had a simple diarrhea for two or three days and lost fifty pounds of body weight, it can be assumed, if there is no other cause for weight loss, that he has lost 5 or 6 gallons of fluid and electrolytes. A five per cent loss in body weight from diarrhea is of no serious consequence because the horse can stand such loss without functional impairment. However, in the same horse a ten per cent loss in body weight in a twenty-four hour period would result in a marked reduction of blood volume, a failing cardiac output, acidosis, severe electrolyte loss, and reduced renal function. These acute syndromes require immediate intravenous therapy to sustain life. Acute gastroenteritis often causes an extremely rapid loss of fluid and electrolytes and will require 10 to 15 liters of intravenous fluids and electrolytes to prevent shock and death.

Each horse is an individual problem and should be treated individually. Laboratory studies can be extremely beneficial. Here caution is advised as there are several causes for the receipt of misleading test results, the most common being hemolysis of blood. This can be caused by heat as happens when samples are left for a day in a warm automobile. Dirty syringes and tubes along with poor laboratory technic and faulty reagents also merit consideration. This discrepancy is noted most frequently in the case of potassium estimations. If water or alcohol is present in the syringe or a detergent solution is used for storing needles, some very inaccurate calcium readings will result. The use of a 20 gauge needle is apt to traumatize cells. Less cell damage is caused by using a 16 gauge needle. However, we have often observed that laboratory determinations also frequently show great variation. Some years ago, I had a client who became dissatisfied with the laboratory procedures we were using. From twenty-two horses, blood was taken for cell count and chemical analysis. We split these samples three ways and sent them to three different laboratories. There were some astonishing discrepancies in the levels reported back to us! For this reason, the clinician still has to use his clinical judgment on each horse and regard laboratory results only as an aid towards diagnosis and treatment. This requires a comprehensive knowledge of the certain basic physiological levels for horses.

The total body water content is estimated to be 70 per cent of the body weight in a thousand pound horse. There would be 700 pounds of water, or about 85 gallons, or 320 liters. This volume is estimated to be distributed between the intracellular and the extracellular compartments in a 1:1 ratio, or approxi-

mately 160 liters of intracellular and 160 liters extracellular water. The blood volume is estimated to be 8 per cent of the body weight or 35 to 40 liters. The plasma volume in a thousand pound horse is 21 to 24 liters.

The most common manner of determining the state of hydration is by hematocrit determination. The normal hematocrit of the adult thoroughbred horse's blood is 40-45 per cent; yearlings 37-40 per cent; weanlings 35-37 per cent. The chart below is a general guide to initial fluid volume requirements.

HEMATOCRIT	INTRAVASCULAR	
	CELLS	PLASMA
40%	16,000 cc	24,000 cc
50%	16,000 cc	20,000 cc
60%	16,000 cc	16,000 cc
70%	16,000 cc	12,000 cc
80%	16,000 cc	8,000 cc
90%	16,000 cc	4,000 cc
100%	16,000 cc	0 cc

When there is a rise in the hematocrit there is a rapid movement of fluids from the extracellular or interstitial compartments in the vascular system. For example, a horse with severe diarrhea with a hematocrit of 70 per cent has lost 12 liters of plasma volume and is in a state of shock and needs immediate intravenous therapy. However, the hematocrit levels do not reflect the total body loss of fluid and electrolyte. Here, it is well to remember that when the laboratory results are received the figures have to be translated into terms that are usable, because the electrolyte levels of the plasma only have been given.

In addition to a hematocrit determination, certain other laboratory procedures are helpful. The serum levels of electrolytes can be studied in many laboratories which have flame-photometers. The following normal ranges are a general guide for electrolyte levels.

BASE (Cations)										
						MILLIEQUIVALENTS				
MILLIGRAMS PER 100 cc						PER 1000 cc				
Sodium	320	to	350	X	.435	139	to	152		
Potassium	18	to	25	X	.256	4.1	to	5.6		
Calcium	12.5	to	13.5	X	.5	6.25	to	6.75		
Magnesium	2	to	4	X	.82	1.64	to	3.28		
ACID (Anions)										
Chlorides	340	to	385	X	.28	95	to	107.		

In the past, nearly all the laboratory results have been stated in terms of milligrams per cent of blood, so if one multiplies that figure by ten, converting from the 100 cc to a thousand times the valence, divided by the atomic weight, the result will be expressed in milliequivalents per liter. For example, in the horse, sodium normally runs from 320 to 350 milligrams per cent, or 139 to 152 milliequivalents. Every veterinarian then should know how to interpret these findings in order to estimate electrolyte replacement.

All this, then, forms a basis for the proposed treatment of many conditions and the therapy depends upon the illness and the degree of dehydration, determination of the type of electrolyte imbalance and organic function.

One should think in terms of deficits and excesses of fluids and electrolytes. On examination of the horse, one can determine dehydration from the appearance of the eyes, the amount of saliva, the elasticity of the skin, the color of visible mucous membranes, and body weight. The normal daily intake of water which varies from 2 to 8 gallons, and the disease from which the horse is suffering must also be considered.

In replacement therapy, there are four main types of solutions (a) glucose in water, (b) electrolytes as balanced or polyionic solutions, alone or in combination with dextrose, (c) amino acid solutions, with or without dextrose, alcohol, etc., (d) blood, oxalated or citrated; plasma or plasma expanders. Commercial solutions have the advantage of being sterile and pyrogen-free.

Glucose solution without electrolytes is a source of calories and tends to decrease certain detrimental effects of starvation, ketosis and destruction of protein. It also tends to prevent the wastage of electrolytes which are being excreted in the urine and it may be used to replace the abnormal loss of water which occurs in many febrile diseases. It should be noted, however, that if the percentage of dextrose in the solution is too high (50 per cent), the horse will be further dehydrated because glucose is a diuretic. There is a tendency to administer this solution at too fast a rate. Five per cent dextrose is isotonic and best for our purpose. An isotonic solution when given intravenously will neither increase nor decrease the size of the size of the cell because it has the same osmotic pressure as the cells.

There are many commercially available solutions containing electrolytes in water. Isotonic, normal or physiological saline is one of the most commonly employed solutions. In the horse, the normal chloride level in the blood lies between 340 and 385 mg or 95 to 107 mEq. The sodium level varies from 320 to 350 mg or 139 to 152 mEq. Isotonic saline has 154 MEq of sodium and 154 mEq of chloride per liter. In a case where there is an excess of

chlorides and isotonic saline is given, the horse receives more chlorides than are normally present in his blood. For a long time, I used lactate Ringer's solution because the lactate was converted to bicarbonate. The sodium chloride content is basic; the additional sodium is required to raise the existing sodium to a normal level. The lactate is converted to metabolism, without the excess chlorides. In addition this solution contains a small amount of calcium and potassium. Recently there have appeared, so-called balanced electrolyte solutions, Polysol and Ionosol, but again these are low in calcium. The most common discrepancy in commercial solution is a lack of calcium. In almost all race horses, except the very young, the calcium content is said to be between 12.5 and 13.5 milligrams per cent, or 6.5 to 6.8 milliequivalents of calcium. On checking the formula on the label it is noted that the figures apply to the human in which the calcium content is about 5 milliequivalents. Therefore, to be polyionic for horses, additional calcium to the extent of 1.5 to 2.8 milliequivalents per liter is required.

Various commercial preparations of protein such as "protein hydrolysates", "enzyme digests", are available for parenteral use. These are available in various combinations of glucose, saline, alcohol, vitamins, and so forth. Amino acid solutions serve to supply the body with an intake of nitrogen essential for growth, cellular repair and wound healing. They are a source of energy and are a hydration solution. They are indicated after the third or fourth day of illness. Two to four liters, two or three times a day are indicated in most conditions, particularly where horses refuse to eat. The rate of administration should be slow. These solutions can be administered singularly or with the multiple electrolyte-dextrose solutions.

Blood is indicated in treatment of anemia, acute and chronic hemorrhage, shock, and in conditions where it is necessary to increase blood volume and cell count. I have not had sufficient experience with blood transfusions and feel some apprehension about transfusion reactions. Blood plasma is indicated in hypo-proteinemia and shock. The lack of available donors for and proper handling storage facilities by the practitioners have reduced this approach to therapy. Dextran may be used as a plasma substitute. The intravenous injection of two to four liters of 6 per cent solution in a 1000 pound horse is indicated in shock.

In using any of these fluids we refer to our chart on "The Initial Fluid Volume Requirements". There is commonly a recurring fluid and electrolyte deficit in these cases, depending on the severity and duration, which requires a continuous infusion or infusions, 3 to 4 times per day. It is further suggested that clinical observation of visible mucous membranes, renal function, heart

rate, body weight, etc. may dictate that additional quantities are needed. Oral intake of less than 4 to 5 gallons of water per day will require stomach tube administration of water and electrolytes. One important point to keep in mind is that in diarrhea, a horse cannot retain water in his system unless sufficient electrolytes are present. This applies to both oral and intravenous administration of water.

One other very important aspect of therapy is the time which has elapsed from the first sign of illness to the time the trainer contacts the practitioner. This is particularly true in acute gastroenteritis in that the time lapse is a major item in survival. Our most recent case was discovered by the trainer at 12:00 P.M. and he called us at 4:00 P.M. Upon observation, the horse was extremely depressed, refused food and water; the visible mucous membranes were cyanotic, the heart rate 68, the temperature 97, and rapid respiration was noted. The attendant stated the horse had not voided urine in several hours. Physical evidence of severe diarrhea was present on the walls of the stall and in the bedding. A blood specimen was taken and a four liter infusion of 5 per cent dextrose was started followed by an 8 liter infusion of balanced electrolytes with dextrose. Two ounces of oral electrolytes were added to the drinking water, and a sedative, along with oral and intramuscular antibiotics were administered. There was a marked improvement in the color of the visible mucous membranes, in heart action, breathing, and general appearance. Treatment was continued 3 times daily for 4 days. A total of 48 liters of balanced electrolytes with dextrose and vitamins were infused; 2 pounds of oral electrolytes were used; and 8 liters of amino acid solution were infused. On the fifth day the horse began to eat and recovered uneventfully.

In fluid therapy, the object is to correct dehydration or overhydration, to make up water or other imbalance arising from intestinal, renal or hepatic disease, to correct acidosis or alkalosis, to stimulate certain organic functions and to make good the deficiencies. In my practice, extensive fluid therapy has markedly increased my percentage of recovery and I feel that treatment, time and drug costs are justified.

"PRACTICE IN THE HOLLYWOOD FILM INDUSTRY"

C. H. Reid

EDITOR'S NOTE: As mentioned in the Foreward it was not possible to transcribe and at the same time, do justice to this presentation. Some idea of the humorous manner in which this paper was presented can be gathered from the opening sentence of the talk:— "I've got so much to say, and so many slides to show, and I'm going to do it if I am the last man left in the room — if you get bored and walk out somewhere along the line, you won't offend me a bit".

The speaker began by describing the type of practice in which he was engaged and went on to say that he had his share of critics and on occasion he encountered some "nasty" competition including those to whom the dollar sign means more than good ethics.

With the aid of black and white photographs and colored slides, Dr. Reid described briefly the making of motion pictures and gave a list of twenty different species of animals, including wild ones, which he had occasion to handle over the years. Twenty-five different categories of horses have been employed in the industry and the speaker then enlarged on the chariot horses used in the motion picture, "Ben Hur".

An interesting feature was Dr. Reid's visual and oral description of the different types of vehicles, harness and other equipment necessary for "period" pictures. He stressed the authenticity of the material used and mentioned that a large amount of money was tied up in these properties which may be used only occasionally over a period of years.

The speaker went on to describe some of the common ailments of "picture" horses and some unusual accidents that have happened. He also pointed out the problem encountered when animals are moved to different locations: stabling, altitude, terrain, etc. all exerting an effect on the animals.

Dr. Reid also mentioned methods of procuring special effects and vividly described some of the humorous situations that have arisen during his years of work in the motion picture industry.

Of special and topical interest to the audience were the speaker's references to some of the stars, (both animal and

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human) which are featured on television as well as the screens of the movie houses.

In more serious vein, Dr. Reid pointed out that all safeguards are taken to ensure that no animal is exposed to unnecessary hazards and that a veterinarian as well as a humane officer are on the set when the possibility exists that an emergency could arise.

THOROUGHBRED PRACTICE IN THE TROPICS

WILLYS E. LORD

The advances in transportation that have come about in the last few years have resulted in changes in the role of the veterinarian. This is especially true in race horse work where international transport of such animals is a daily occurrence. We no longer have days or weeks elapsing during movement from one part of the world to another, now it is but a few hours; therefore, we must be prepared to recognize exotic diseases in the incubation stage as well as in the advanced forms.

While advances are being made daily in the automation of industry, we in the veterinary profession have not reached the point where we can put an animal into an IBM machine and receive a punched card diagnosis. We still have to rely on human professional judgment in order to make a diagnosis.

I imagine that some of you wonder, from the title of this presentation, how a veterinarian in the Army could be on active duty and still be doing thoroughbred work at the same time. I recently returned from Panama where for a little over four years I was assigned to the Panama Government as Chief of the Army Mission. In addition to being a Veterinary Advisor to the Ministers of Agriculture and Public Health, at least fifty percent of my time was occupied working with the Guardia National or Panama Army and Police Force.

The Government of Panama owns and operates the race track; therefore, I was called on for advice concerning a new race track that was being planned at the time of my arrival.

My first impression of racing in Panama was not too favorable. When I attended the races for the first time at the old track the crowd shouted, following one of the races in which a horse ran completely off-form, "Hernandez, Hernandez". I asked the meaning of this and was told that, "He is one of the local veterinarians", and they knew he had something to do with this — such was the reputation of the veterinary profession in Panama a few years ago.

The contacts with horse owners, trainers, and veterinarians, while working with the horses, made my official duties much easier as the President of Panama, Minister of Foreign Affairs, Minister of Finance, and numerous other officials, were all horse owners. Official as well as horse problems were discussed while at the track. Doors were opened that would have been more difficult through official channels.

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As previously stated the Government of Panama was planning a new racing plant when I arrived, so I was in on some of the planning and arranged to have a Veterinary Clinic constructed that compares favorably to any that I have seen anywhere in the world. The track was built at a cost of 5½ million dollars and leaves little to be desired.

The Veterinary Service that we set up is somewhat different from that in this country, in that the Government paid the veterinarians on a monthly basis and the horsemen were charged only for medicine and materials. Two Panamanian veterinarians, one from Cornell and the other a Colombian graduate, worked with me. Horses were brought to the clinic at specified hours for treatment while care outside regular clinic hours was on a fee basis.

Saliva and urine testing was instituted shortly after opening the new track. A Tulane graduate chemist working with Eastman Kodak, was sent to Miami to work with Dr. Bellows, where he was given excellent training. Of the first two positive cases, one was owned by a relative of the President of the country, and the other by one of the cabinet ministers; however, sanctions were applied and from then on the problems were eased.

In conjunction with the track work, visits were made to breeding establishments and advice furnished on management, feeding, sanitation, etc. There are six major breeders located from sea level to an altitude of 6,800 feet — resulting in a variety of climates and conditions.

The native pasture grasses are elephant, para and jaragua, lush growing but of little nutritive value. I have seen grass tall enough to hide a horse and yet the animals showed nutritive deficiencies. On one farm of 40 hectares, which is 100 acres, there was insufficient pasture for 35 animals for year-round grazing. I introduced Pangola Grass from Florida along with pasture management and fertilizing; now that same 40 hectares pastures the horses and 65 head of cattle.

There were some erroneous ideas about horse management, one being that heat and sun were bad for horses. They would be stabled during the day and pastured at night only, resulting in a condition where the hair would come off in large patches due to avitaminosis. The practice also contributed to mineral deficiencies, which I shall elaborate on later. The conditions clear up by bringing the animals into the stables for a few hours only during the hottest part of the day.

The government also buys the horses, financing and reselling to individuals. I made several trips to Argentina, Chile, Peru, and Brazil to advise on buying animals for racing and breeding. I also purchased bulls from Mexico for use in the bull fights in Panama.

I will discuss a few of the diseases encountered in Central and South America that are normally not seen in every day practice here. We should guard against new diseases being introduced into the United States through negligence on the part of any veterinarian who fails to recognize these diseases. With flying time from country to country being reduced to a few hours, we may see diseases not previously seen.

Piroplasmosis was first reported in Panama by Darling in 1913. It is hard to confirm this disease by laboratory methods, but the symptoms are unmistakable. Those of you from Florida or the southern region will have seen tick fever in cattle. The horse presents a similar picture. An acute outbreak is characterized by temperatures ranging from 105 to 107 degrees. There is marked depression, inappetence and jaundice. Many cases will show hemoglobinuria, along with marked anemia. There is a rapid loss of weight, the gait is uncertain, resembling that of a heavily parasitized horse.

This condition is fairly common in native horses throughout Central and South America; however, I have seen several cases in race horses in Panama. It does not occur often in these animals because of the manner in which they are maintained.

During the acute stage of the disease, which lasts for five days, the intracorpuscular parasites are easier to find than when the condition becomes chronic. Where the parasites cannot be found, blood counts and bilirubin determinations are helpful. In fact, we ran routine blood studies on these animals. I recall one animal whose red cell count dropped from 9 million to 6 million in seven days, and to 3 million in sixteen days.

For treatment, large doses of tetracyclines were used with variable results. Good results were obtained with Acaprin, a Bayer product from Germany. Trypan Blue has been recommended, but we were unable to achieve the advertised results.

In very acute cases, deaths occurred within 24 to 48 hours. The average duration is about 7 to 14 days, with a convalescent period up to six months. During the convalescent period, the animals are very susceptible to pneumonia, enteritis, and other secondary infections.

Where treatment is ineffective, necropsy reveals a large, soft, brown spleen; thin, watery blood, and jaundice of all the tissues. The liver is swollen and of a rather yellowish color.

Prevention through the elimination of ticks and other biting insects is fairly effective, however, in some countries the disease has been transmitted by mechanical means such as the Vampire Bat.

Trypanosomiasis is another problem. This term covers a group of diseases, both animal and human. The first published record of

the disease was in 1841. The syndrome in horses is known in various parts of the world as surra, nagana, tsetse fly disease, dourine. In South and Central America it is known as mal de carderas or murrina.

In the Americas it was first reported in Colombia and Venezuela. In Central and South America, Trypanosoma hippicum was the causal agent, while in Europe and Africa, T. equinum and T. equiperdum have been encountered. Trypansoma vivax was fairly common in cattle but did not affect horses.

The clinical picture is marked by fever of a rather irregular character; progressive emaciation, anemia and faint icterus. The hair coat is rough and frequently there is edema of the scrotum or lower abdomen. Generally, there is weakness of the hindquarters, and later animals show incoordination, however, incoordination has been noticed as the first symptom. The resemblance to wobbles is great so one should be on the lookout for this symptom in imported horses. The clinical picture is presumptive evidence of the disease, but laboratory tests are necessary to confirm the diagnosis and differentiate it from other similar diseases.

In thick blood smears, the organism is fairly easy to find. Thin blood smears were not of sufficient concentration to detect the organism. Laboratory animal injections, using both mice and guinea pigs, were carried out. In mice, the disease is always fatal; in guinea pigs it is not always fatal, and the blood teems with trypanosomes in those animals that survive for any length of time.

A satisfactory homologous antigen has not been readily prepared; however, it has been found that where Trypanosoma equiperdum antigen was used in the complement fixation procedure, there was a correlation between infection and positive serological findings.

Animals other than horses are affected by Trypanosoma cruzi, cattle being an important reservoir of the infection. It is believed to be non-infectious to man. Infection can be transmitted by mechanical means and a frequent means of transmission is the Vampire Bat, strictly a mechanical procedure, but at least 50 per cent of the cases we saw could be traced to bites from Vampire Bats. By permitting biting flies to actually walk through the blood taken from acute cases in guinea pigs, it is possible to infect horses. In some areas we found it necessary to either keep lights burning in the stables or screen the stables to protect the animals from Vampire Bats.

For treatment, we used potassium or sodium antimony tartrate, and the results were fairly good. In Africa, in the trypanosome areas, they find this very effective. Bayer 205, or Naganol, in 5 gm doses intravenously, gave very good results; in fact, we used this as a prophylactic treatment, in native animals, espe-

cially, in areas where the disease had a high incidence, and during the rainy season. We gave four injections, two months apart, and found this was the best means of controlling the disease in those areas.

Two British drugs, Ethylene Bromide and Antrycide are effective in cattle but not in horses.

Some work was done on viral encephalitis. This disease was first recognized in 1936 in Panama and has been there off and on ever since. A small outbreak took place in 1945. In 1948 there was an outbreak in the central provinces where more than 500 animals died in a short time. Then the disease disappeared. No more was seen of it until 1955, when one confirmed case was detected in a riding stable.

The next outbreak was in 1958 when an outbreak of trypanosomiasis was being handled. One of the local cowboys reported that some of the horses on a neighboring farm were, as he described them, "bustante loco" which means "completely crazy". So, we started doing some work and were able to isolate the Eastern strain. Then Major Tom Murname, a laboratory veterinarian assigned to the mission with me, branched out into a full scale investigation of the disease, ran studies on insects, birds, native animals, rodents, and took blood for study from innumerable horses. Approximately 60 human cases were also studied. A paper on this outbreak has been submitted by Major Murname for publication.

During this investigation a total of 23,069 females, belonging to 56 different species of insects were trapped. The Mansonia was by far the most abundant species. There were approximately 16,000 of them. In addition, 4,000 Culex, 752 Anopheles, and so on down the line.

We caught and bled 68 bats of various species; 17 spiny rats; 13 opossums; numerous field mice, and so forth.

Very high titers were found in some of the birds examined. We also found high titers in the insects and in some of the humans; and approximately 75 per cent of the horses in this particular area were shown to have been exposed to the infection.

Vesicular Stomatitis was another condition encountered. The disease is not serious in itself, however, if an outbreak occurs in cattle the economic factors present a problem until Foot and Mouth has been eliminated as the diagnosis. Panama is very conscious of the prevention of Foot and Mouth Disease, as all countries to the south are considered infected. I have seen 28 of 36 horses on one farm with Vesicular Stomatitis, all showing symptoms within 24 hours. Treatment is local, however, the animals lose weight rapidly due to difficulty in eating. They are off feed for about 5 days. It takes a little time to get them back in shape and it does hit the owner in the pocketbook.

Torsalo caused by Dermatobia Hominis, a grub type infection, similar to cattle grub, caused some difficulty in horses. It differs from cattle grub in that the fly lays the larvae directly on the skin. The larvae penetrate the skin and live in that spot for 40 to 60 days. The first evidence is a small hole exuding serum, it resembles a stab wound or may be confused with a bullet wound such as is caused by a .22 rifle. Antibiotics are ineffective in the treatment of the wound unless the grub is removed mechanically or killed by insecticides. In some areas horses and especially cattle may have hundreds of Torsalo — the losses in value of hides due to the holes are substantial in addition to the loss of weight, secondary infections that may cause deaths, etc. Humans are also affected. The condition is controlled by regular bathing or spraying with insecticide at three-week intervals. Some of the newer systemic phosphorus compounds show promise of even better control.

Leptospirosis and African Horse Sickness were not encountered nor was infectious anemia diagnosed in Panama.

Internal parasites are a constant problem.

For strongyles and ascarids at breeding farms, we used low level treatment and supplemented that by periodic treatment using a mixture of phenothiazine and piperazine by stomach tube. In training, the low level treatment is not used. One of the speakers yesterday remarked about the esophagus being on the right side of the animal. In one stable, four out of twenty animals had the esophagus on the right side. In these heavily parasitized animals, the R.B.C. would be as low as one million and this was a not uncommon finding especially in the native animals. After worming we used iron dextran and B$_{12}$ for building up the hemoglobin and red cell count.

In addition to strongyles and ascarids, we saw several cases of anoplocephala perfoliata infestation. We treated one animal ten times while I was there, and we still have to find an effective treatment. I imagine those horses are still infected.

It was most surprising not to encounter bots even in imported animals. Cases have been reported from Costa Rica, however, none was found in Panama. I can't explain why, but they were not there.

External parasites presented no problems, as they can be controlled by the newer insecticides.

A type of diarrhea presented quite a problem; it was finally traced to a cilia type organism. The animals presented the usual symptoms found in diarrhea. The stalls would be "painted" tail high as with a pressure sprayer. Mortality approached 100 per cent. Considerable work was done and the organism was found in rat and mice feces and urine. The disease was controlled by rat-

proofing feed storage areas and protecting the feed racks and buckets by putting metal sheets on the walls to prevent access to them by rats and mice. Just prior to my leaving there, we started using Camoquin, a human anti-malarial drug, and obtained very good results.

A big problem, and it is common, is non-sweating. It is a condition sometimes called dry-coat in which the animal sweats little or not at all. I first encountered this condition in India in 1942. Animals in this condition perform poorly and, if racing is continued, may drop dead at the finish line. We tried many treatments including thyroid extract, iodine, electrolytes, adrenal contex, and steroid compounds, without results.

The only treatment or prevention that we found to be effective was moving the animal to a cool climate or to air condition the stalls. We had about 20 animals in air conditioned stalls. Affected animals that haven't sweated for six months will begin sweating after exposure to cold temperatures, in as little as four days. This is a problem in tropical medicine still to be solved.

Mineral deficiencies were a great problem. I remember Dr. Guard used to say that mineral deficiencies had a lot to do with bowed tendons and breakdowns, popped knees, bad ankles, and I didn't believe him until I got to Panama. The first year I was there some 28 local-bred horses came on the tracks for the first time, and in six months not one of those was still running. I can attribute almost all of the breakdowns to mineral deficiency.

We started a feeding program, supplementing the rations with minerals, but one mistake was made. We advised bone meal. We found in our blood studies that the calcium and phosphorus were completely out of balance. The bone meal prepared from locally slaughtered animals showed the same imbalance, so in some cases the condition was compounded rather than helped. A switch was made to imported bone meal and trace minerals were added. Today we have no specific problems and mineral deficiency in animals that are properly cared for.

Dr. Proctor's operation for bowed tendons was tried; however, the results were not too good; the animal usually broke down in the other leg.

In the treatment of coronary injuries, especially suppurative coronitis, I used an unorthodox procedure that may cause some of you to shudder. Instead of peeling down the hoof wall, I would give a local anesthetic and then using an electric drill, with a $\frac{3}{8}$ inch bit (sterilized), provide bottom drainage by drilling straight down through the sole. A drain with some antibiotic was inserted and changed as needed. Healing was rapid and return to racing was prompt.

Umbilical hernias were repaired using Vetafil; however, I found out since returning that it is not recommended by some for

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buried sutures. The results were excellent and horses were racing within three to four months following surgery at two years.

We used firing and blistering for various lamenesses, with the usual results. Bucked shins responded best to infiltration over the periosteum of prednisolone or hydrocortisone. Results were excellent; in fact, 48 hours after treatment one animal won a two year old classic. It may have to be repeated, but I have observed no adverse results.

I find the plastic surgical drapes with adhesives are ideal for horse practice. They are available in any of the surgical supply houses, as they are commonly used in human surgery. The incision is made directly through the drape. It stays in position even if the animal should move. It is especially good when local anesthesia is used and we seldom used general anesthesia.

In conclusion, I'd like to mention my three pet peeves:

One is where the veterinarian going out to treat a horse takes out a vial of procaine tablets, pops one on to his hand, picks it up with the syringe and adds tap water, or may take some out of a bottle. He then starts injecting around the joint of a horse. I shudder every time I see that.

Dr. Walker yesterday mentioned a dental anesthetic. It is very satisfactory but I prefer to use the dental type of syringe — a break-away type of syringe, into which is placed the cartridge. It is available in 1.1 and 2.2 cc ampules. You can block an ankle or knee with 5 to 10 ampules. The 2.2 size costs about 2½ cents each, so the cost for blocking a knee or ankle is 25 cents at the most and it looks much more professional than the old procaine pill and water procedure.

Another pet peeve of mine is the so-called ethical drug house! The container-labels have all over them in Spanish or English, "Sales to graduate veterinarians only." Yet, they are seen in every feed store, corner grocery store, and drug store. I assure you that the veterinarians of those countries take a dim view of having ethics in veterinary medicine apply only to the United States.

My last pet peeve concerns veterinarians visiting in foreign countries. They come in as visitors or on a consultant basis to a trainer or breeder, or to the Government. In many cases they do not contact the local veterinary society or the head veterinarian of the Government. The latter learns he was there, through talking to somebody or by reading about it in the paper. It does not lead to better inter-American relations to have people come in without coordinating with the veterinarians of the country visited. This person-to-person diplomacy advocated by the President, I think, can start in the veterinary profession. We have enough trouble in some of these countries without alienating the veterinarians of these countries against the profession in this country.

PANEL ON THE DIARRHEA PROBLEM IN THE HORSE

Moderator: M. B. TEIGLAND

Panel: J. M. MILLER
W. L. ANDERSON
J. E. BURCH

MODERATOR TEIGLAND: As everyone is aware, the diarrhea problem is cropping up in various parts of the country. Depending on the locality, the etiology may be different. It may be the same. Perhaps this panel can straighten our thinking on this particular syndrome.

DR. MILLER: In describing the different manifestations of equine viral arteritis, Dr. E. R. Doll mentions an intestinal form and it is an apparent outbreak of this acute intestinal condition that I wish to discuss, for in the past several years, it has proved to be a perlexing problem in different areas of the West. With reference to equine influenza, Udall's text relates that "In certain years one meets with an intestinal type in the form of a highly fatal gastroenteritis. This should not be confused with a terminal diarrhea."

The symptoms of this condition are acute. Severe scouring comes on suddenly with colicy pains, depression, dehydration and shock. The excreted fluid has a peculiar but characteristic odor. The mucous membranes of the lips and gums become cyanotic. The cyanosis is due to anoxia from hemoconcentration which is secondary to rapid dehydration. In the first stages, or in a milder attack the gums will be intensely red. If the disease progresses and scouring persists, the membranes darken to purplish black. There is severe injection of the scleral vessels. Urine formation is scanty and the coloration is that seen in a case of azoturia. Temperature varies from 103° to 107°. In two cases in older horses, one five, the other six years of age, typical cramp, or hardening of large muscle groups was evident.

In the outbreak at Ruidoso Downs race track in the summer of 1959, most all the animals followed the above pattern of symptoms. This year however, although the number of cases are not as great, we have been some atypical cases. One case developed severe cardiac arhythmia, and another developed encephalitis.

In the former case the animal was responding well to treatment. The scouring and cramping was under control, the strength

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and attitude of the animal had improved and the membranes were returning to a normal pink color when arhythmia was noted. Death occurred five hours later.

Where cyanosis was present, death occurred within four to ten hours in about seventy per cent of the cases — the younger the animal the higher the fatality rate. As mentioned previously, horses up to six years of age were affected but mortality was mostly in horses two years old.

The cause is a multiple factor situation. Five to fourteen days prior to the acute symptoms of gastroenteritis, in ninety per cent of the cases, there was a history of typical influenza-type respiratory infection. The temperature was around 104° to 105° with labored respiration, loss of appetite and depression. Nasal discharge and cough are usually absent at the onset but may develop in two to three days. These cases respond quite readily to antibiotics or sulfonamides and symptomatic therapy. Within three to four days the animal is back on feed and looking fairly well. It is human nature to hurry, so within a week some of these colts are back in training. Of the first five fatal cases which occurred at the track, every animal had been worked hard on the day of death or on the previous day. Two of these cases were checked within minutes of returning from the track and found to have a temperature of 106°. Colicy symptoms and scouring were immediately evident. Leukopenia was present due to the original viral infection. With the lowered resistance over-exertion and fatigue constitute "the straw that breaks the camel's back". Evidently the normal bowel bacteria become pathogenic and acute gastroenteritis results.

Bowel cultures were negative except for E. coli on one specimen. These cultures were from animals which had received oral and systemic antibiotics. Due to the acuteness of the disease the formation of an enterotoxin was suspected but to my knowledge none has been isolated.

On autopsy the tissue changes may be those of acute septicemia or gastroenteritis. The mucosa of the stomach is usually hyperemic or hemorrhagic. Edema is present in the small intestine and small colon. Hyperemia and congestion of the lungs may be present. Petechial hemorrages may occur on the visceral peritoneum of the bowel. In those which died within a short period, tissue changes were absent except for hyperemia of the stomach and small intestine. Tissue changes were however extensive in those surviving for two to three days.

Treatment is far from satisfactory, yet some animals will respond even when in the early stages of circulatory collapse and when cyanosis is evident. The reason for this is unknown.

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In the early summer of 1959, the first five cases showing cynosis died. Then several cases which were expected to die, responded to treatment. Sometimes a relapse would follow apparent progress towards recovery and death would ensue.

Intravenous fluid therapy consists of electrolyte solutions and five per cent dextrose-saline. There were no facilities for carrying out blood analyses but a rule of the thumb which I follow now is one unit of electrolyte solution to three units of five per cent dextrose and saline. Usually four thousand cc of these fluids are given to those showing a response in a twenty-four hour period. I start with 1000 cc of normal electrolytes and every four to six hours give 1000 cc of five per cent dextrose-saline for three injections before repeating the electrolytes.

In severe hemoconcentration one is forced to give large quantities of fluids. An immediate response to the dilution of the blood by the fluids mentioned is obtained even in a horse that is down and in severe distress from anoxia. Usually when the condition has progressed to this stage, the response is only temporary. A continuous drip seems to give better results than does intermittent administration of fluids. Blood transfusions were of no value. In one case, a filly, blood was used from a five year old gelding which one month before had recovered from the acute symptoms. We did not save the filly and the donor developed gastroenteritis the next day and had to be treated all over again. Normal horse serum has given inconsistent results. Gelatin solutions were used but the results could not be assessed.

Penicillin-streptomycin was of no value in my hands. Triple sulfas worked in some cases. Chloromycetin was used in two severe cases and both horses responded well; however, this drug in the amounts required is highly impractical due to the cost. Terramycin in doses of two and one-half grams intravenously twice daily is my choice of therapy at the moment. One theory is that this acute form of gastroenteritis can be caused by treatment with terramycin. Many of these cases never had terramycin previously so this theory is unlikely.

Dypyrone is indicated for the spasmodic colic which occurs and it also helps to control the high temperature. Stress hormones are of value and I prefer ACTH over the cortisone derivatives.

Dr. Peyton of Jen-Sal Laboratories was kind enough to send me a supply of the anti-spasmodic called Jenotone prior to its release on the market. It is very effective in relieving the colicy symptoms, present at the onset, although it is perhaps not specific in stopping the acute diarrhea. Preparations to coat the bowel are important. An initial dose of 8 oz. of corrective mixture with paregoric and 16 oz. of Haver-Lockhart's, Skoursul with electro-

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lytes. Four oz. of each preparation with 1 oz. of neomycin is given every four to six hours. Anti-histamines are of some help in the extreme toxemia.

Parenteral enzymes such as Varizyme and Chymar are indicated for the edema of the bowel.

In cases with myoglobinuria, calcium gluconate, thiamine and anti-histamines are indicated.

Stimulants are used cautiously but to advantage. In animals with extreme exhaustion 10 cc of 25 per cent nikethamide solution are used.

A small amount (heaping teaspoon) of salt in the drinking water is used routinely. If permitted these animals will, on occasion, consume gallons of water. Constant care and logical nursing is vital and cannot be over emphasized. I remember one morning about 3 a.m. having to tell one old trainer that his colt was dying. The colt could not get up, his mouth was dark blue and the palpebral reflex was absent. The old man did not believe me. He followed all of my instructions to the letter and some of his own, I imagine, and the colt lived and has won some races since. A few experiences like that have made me a believer in constant attention to these cases.

Complete rest for at least three weeks after the initial respiratory infection prevents most cases of gastroenteritis. The majority of these animals were futurity colts and a three-week lay-up would have cancelled their chances in the trials. The largest death loss was at this time. It was not long until the situation made believers out of those who tried to substitute for rest and time. Although some horses were periodically sick the death rate subsided.

Over a nine month period last year at three different race tracks in New Mexico about sixty cases of gastroenteritis were seen. Of this group, twenty per cent were of the highly acute form and in this type, the mortality rate was about seventy per cent; however, the largest number of fatalities occurred in the early summer of 1959 when the disease first appeared.

There is no specific vaccine for viral arteritis although I understand that some work is being done along this line. Use of Equine Bacterin, number one, is of great help. Last fall and winter I preached the use of it to all the trainers and owners I knew who would be bringing colts back this year. Many of these colts were vaccinated this year before coming to Ruidoso and we also vaccinated a large number at the track. Although respiratory disorders and typical influenza were still encountered, the incidence of the gastroenteritis complex was at a minimum. I would guess

there were four hundred colts vaccinated and only two cases of gastroenteritis occurred in this group, one of which died. In a group of about one hundred un-vaccinated colts there were at least ten cases of gastroenteritis and three fatalities. I definitely believe that the bacterin affords some immunity against the secondary bowel invaders. Three injections are given into the gluteal muscles; a first injection of 5 cc and on the two succeeding injections, 7 - 10 cc. Stiffness and soreness will occur frequently and occasionally anaphylaxis. A booster injection should be given nine months later.

MODERATOR TEIGLAND: Dr. Anderson is the next speaker.

DR. ANDERSON: Down in Texas we certainly see the same syndrome which Dr. Miller described. Viral arteritis is a condition ever-present in our minds but I believe there are other entities of significance. My equine practice is concerned with the Quarter horse although most all breeds are present in the area and have been treated for the condition just discussed.

Apparently the young animal is more susceptible and I have seen the condition in young foals.

When severe diarrhea occurs, one immediately thinks of the salmonella group of organisms but cultures and smears have failed to reveal the presence of this organism.

In colts, there is an immediate diarrhea, with elevation of temperature, extreme depression, colic, dehydration and if not treated, death. In our particular location, horsemen now realize that treatment must be prompt if the animal is to survive.

As far as environmental conditions and husbandry are concerned, we meet a great variety. The stables and breeding farms vary from good to bad and sanitary to insanitary.

I like to divide this form of diarrhea into three different types; chronic, peracute and acute. The chronic type starts out with no temperature elevation, no depression, no cyanosis and no typical signs of colic, but it does persist for days, weeks and sometimes months. I believe this chronic form may be due to parasites for in these cases a high incidence of strongyles with some strongyloides is evident.

In the acute form, I believe that, at least in some instances, resistant bacteria may complicate the picture. Consideration might also be given in our part of the country to toxicity by chemicals or rather insecticides, for the latter are used routinely. The cotton plant is sprayed throughout the season with phosphorus-containing insecticides. The manufacturers claim, however, that the agents in their products are dissipated or dispersed within a short period of time. We also see the defoliant in the cotton which

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evidently contains arsenious acid and occasionally acute arsenical poisoning is seen.

The case of chronic diarrhea is treated by worming the horse and turning it out to pasture. Several different therapeutic agents have been used but it seems that results are just as good following minimum medication and the turning out to grass.

In the acute case especially, I used blood transfusions routinely, with apparent benefit. Plasma does not seem to be as effective as whole blood, judging by the results obtained from the use of plasma from recovered acute cases.

The blood picture in the acute or peracute diarrhea indicates definite evidence of a viral infection. A typical peracute case has a red cell count of 3800000; hemoglobin level of 7.7; hematocrit of 65 and a sedimentation rate of 98. Such animals are usually dead by the time the laboratory results are received.

Other than blood, electrolytes and 5 per cent dextrose are recommended. Broad spectrum antibiotics such as Terramycin, neomycin and Entromycin (Pitman-Moore) are also of value.

In the early case, mineral oil is given immediately or if severe colic is not present, castor oil may be preferred. Intestinal astringents such as Kramastringo (Fort Dodge), Bismol preparations or a mixture of equal parts of paregoric, Bisilad (Central) and Hybephen (Massengill) are of help. The dose of the latter mixture is one ounce every two hours for a weanling and two ounces for a horse weighing about 1000 lbs. Jenotone is of value but I do not think it stops the diarrhea although it makes the horse more comfortable.

If the animal is not too toxic, very small doses of Promazine are sometimes used. It is contraindicated, I believe, when the animals show much toxicity.

With regard to sequelae, enlarged infected joints are seen. Cultural examination from one such lesion revealed no growth. Laminitis, icterus and a failure to gain weight are also encountered.

Prophylaxis on a herd basis consists in turning the unaffected animals out to pasture. They should not be overfed or pushed too rapidly for weight gain. A sound, well-planned worming program is indicated. It is not known if parasites can actually cause the condition but there is certainly a possibility that they may initiate the diarrhea and permit a virus or bacteria to play a role in the syndrome.

In some stables I have been using mixed bacterins for several years. These stables have about 400 horses and I recall seeing only one case of this particular type of diarrhea during this period,

but it must be admitted that most of these horses are four years old, or older.

Attention to personal clothing including footwear is mandatory. Disposable syringes should be used whenever possible and instruments should be autoclaved. Newly introduced animals should be isolated from the remainder of the horses for at least three or four weeks.

It is very important to avoid the promiscuous use of antibiotics, especially penicillin and streptomycin. Overstocking should be eliminated, for example, many breeders have insufficient room to handle the number of horses present on these small farms.

Particular attention should be paid to animals which have recently undergone stress, for this type of diarrhea occurs immediately after shipping, worming, sudden change of environmental temperature and infectious diseases such as strangles.

MODERATOR TEIGLAND: Dr. Burch is the next speaker.

DR. BURCH: It would appear from the papers just read that the cause of this diarrhea complex varies. I first encountered the acute type as a sequel to shipping fever some years ago. At that time, I was convinced that the cause was a change in the bacterial flora following terramycin therapy. Blood studies indicate, however, that a virus is the probable cause.

A number of pathogens including Proteus, Salmonella and Hemolytic Coli, have been cultured from the gastro-intestinal secretion. There is a question, however, as to their pathogenicity, because the normal bacterial flora of the equine intestinal tract has not yet been established. In my opinion, Salmonella, Proteus and other pathogens incriminated are opportunists which produce many of the enterotoxins seen in conjunction with primary viral infection. Their presence is associated with the acute phase.

Attention should be directed to the use of a bactericidal agent which will control these opportunists. Simultaneously the critical loss of fluids must be corrected. Electrolyte depletion must be prevented while agents should be used which will reduce the hypermotility of the intestinal tract, the associated pain and the depletion of vitamins especially those of the water-soluble group. A group of protein replacement is indicated later.

All the known therapeutic antibiotics and sulfonamides have been tried with varying degrees of success.

As already mentioned, my first experience of this entity followed the use of Terramycin for shipping fever. A typical case would show a temperature of 104 or 105 degrees, dehydration and all the signs of a stress reaction. A marked improvement was usually evident within 24-28 hours. The horse would linger along

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for two or three days and then relapse showing signs of acute gastroenteritis. I have never seen the mass outbreak or contagious syndrome as encountered in New Mexico and South Carolina. My experience has been with the individual case.

To prevent dehydration, it is essential to have some laboratory work carried out on the blood. It is advisable to administer 5 per cent glucose in half strength saline to ensure that the horse receives sufficient quantity to stimulate kidney function. Following fluid therapy, the improvement in these acute cases is truly amazing. The character of the mucosa will indicate whether improvement is taking place especially since a quantity of fluid from 2 to 10 or 12 liters may have to be given before urine in voided and improvement is shown in the visible membranes. The degree of toxemia usually will dictate the quantity of fluids to be replaced.

Following this, a switch is made to electrolyte solutions, since in diarrhea there is a loss of electrolytes in near proportion to the levels in the same plasma. In addition there is interference with the resorption of water and electrolytes especially sodium and potassium, from the intestinal tract. From 4-6 liters, 3 or 4 times daily is considered a logical dose, the commonly employed 1 or 2 liters per day being quite insufficient. It may be advisable to consider administration in the form of a continuous drip.

The horse with diarrhea will usually drink copious amounts of water. It has been my experience on being called to such cases that electrolytes should be added to all the water that the horse will consume. At the moment, I favor Eltras (Haver-Lockhart) as an electrolyte mixture although the exact proportions of electrolytes are not known.

Even though there may be a delay in the receipt of the laboratory reports, the information will help in planning the requirements for subsequent daily treatment if the horse survives that length of time.

A hematocrit test should be carried out. I believe I mentioned that I lived under the misapprehension that if the hematocrit was 70, the loss of fluid from the blood would approximate only 12 liters. The matter of losing some 80 liters of extracellular fluid was overlooked! This large quantity of fluid could hardly be infused intravenously, but the primary concern is to avoid the shock syndrome and acute acidosis encountered at this reading. It is imperative then to use fluids and the visible results are dramatic.

Promazine and Jenotone have been mentioned by other members of the panel. Phenobarbital is also useful. One commercial product contains methyl-scopolamine and is administered by dose syringe or stomach tube or by placing it in the drinking water.

Entromycin, four ounces, twice daily along with the concentrated kaolin-pectin solution and oral electrolytes is to be ad-

ministered. It is obvious from the pathologist's report that there are frequent secondary invaders in this diarrheic syndrome. In my first case, the report was to the effect that the case was one of idiopathic gangrene of the ileocecal valve with the comment that the pathologist had never seen a three-foot specimen that showed such total destruction of mucosa and musculature with pseudomembranous formation.

Visible evidence of toxemia, probably enterotoxemia is present in many cases. Prognosis depends largely on the length of time the animals have been affected and the prompt total therapy administered.

I believe the time is ripe for discussion from the floor.

CHAIRMAN TEIGLAND: Has anybody a question for the panel?

DR. LANG: I can never understand why water is given orally in the treatment of diarrhea. I realize that dehydration is present, but surely by giving water orally, the condition will be aggravated.

DR. BURCH: That question poses a long-standing controversy about fluids. If water is restricted for 24 hours in these cases, the mortality will be much greater. Water by mouth does not pass through the gastrointestinal tract as such because it is absorbed. I believe that the worst that can happen is the increased spasticity of the tract resulting from the consumption in any material by mouth. Water is necessary and I no longer withhold it especially since I now add electrolytes to it.

DR. CHASSELS: What volume of water do you give by stomach tube?

DR. BURCH: I do not give any water when I first start to treat the case.

DR. CHASSELS: What volume of electrolyte-kaolin mixture is used and do you give this by stomach tube?

DR. BURCH: Thirty-two to sixty-four ounces with electrolytes is the initial dose. This contains Entromycin or Biosol.

QUESTION: Did you encounter any involvement of the eighth cranial nerve when using Biosol?

DR. BURCH: No, although several practitioners have experienced this sequel.

DR. WISEMAN: Has any member of the panel had success following the use of Diathal?

DR. BURCH: I had no success with it and discontinued using it.

QUESTION: Could Lentin or Carbachol be used to clear the digestive tract at the commencement of treatment?

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DR. MILLER: They purge themselves! A colleague once gave a large dose of Istizin to a typical case and the animal survived despite the considerable distress caused by the drug. Frankly, I could not employ such treatment.

DR. COSGROVE: In Ireland we do have a viral influenza, similar to that described by Dr. Miller. We are inclined to treat these febrile conditions with broad-spectrum antibiotics, however in doing so, the E. Coli level is lowered and the staphylococci become preponderant, leading to severe gastroenteritis and sloughing of the mucosa. We find that oral penicillin gives good results against this staphylococcus. We also use fluid and electrolyte therapy as described by Dr. Burch.

DR. CHASSELS: Dr. Anderson, I just do not agree with your idea of turning those cases out to pasture. Are you not going to spread infection all over the place?

DR. ANDERSON: That may be true, but we are inclined to meet this condition in congested areas or stables where ventilation and sanitation is not of the best. It has been our procedure to turn these cases out. Of course, the Texas sunlight should soon sterilize the infected pastures!

DR. O'DEA: I think the members might be interested to know that where the bacterial flora has been destroyed, Dr. Cosgrove has to re-inoculate the bowel with normal horse feces.

DR. COSGROVE: Normal feces preferably from a farm other than the one where the horse is being treated.

DR. KING: I wanted to ask Dr. Anderson if he finds a difference in virus between the various farms affected. Also, in his experience what is the age of the youngest animal affected?

DR. ANDERSON: I have seen this condition in foals two or three days old. On one occasion. it started out like an apparent dietary scours, but there was a progressive rise in temperature, depression, congestion, labored respiration and colic. Some difference in virulence is noted and the annual incidence varies from place to place. I have also seen it in backyard stables where maybe one or two horses are present. In these cases, a common finding is that the owner is accustomed to using penicillin on his horses. Perhaps this has something to do with the variation in virulence.

DR. LANG: I recollect an incident about twenty years ago where a mare had a new-born foal which came down with diarrhea and died very quickly. After that, every foal on that farm developed diarrhea. Those over two weeks of age recovered. You could almost set your watch in that when these foals were 48 hours old they would develop diarrhea. A few that did survive developed

joint infection. I had at that time, heard of only one similar outbreak in Central Kentucky.

DR. ZEMJANIS: I was wondering if anyone has observed in foals a type of diarrhea which is very profuse and resists all treatment? The foals grow very well, are slick but start diarrhea at two weeks of age and this persists for about two months. All attempts to isolate the causal organism have failed. The mares are normal.

MODERATOR TEIGLAND: Has anyone comments on this problem?

DR. LANG: Are you sure that this is not salmonellosis? What of the carrier state of this condition?

MODERATOR TEIGLAND: They have not been able to isolate any organism.

DR. ZEMJANIS: Cultures have been attempted from treated as well as non-treated animals. The amazing point is that they do well. The hair coat is slick and they grow like any other colt.

DR. RILEY: I would like to express agreement with Dr. Zemjanis. We have this trouble in Michigan in foals, weanlings and yearlings and even in some two-year-olds. We have not been able to stop the diarrhea either. On culture we have obtained Salmonella newport but do not think this is of any great significance, however, as Dr. Burch has said, we do not know the normal flora in the horse. Chloromycetin which normally would be specific for Salmonella does not help the condition. Symptomatic treatment will help but as soon as they are taken off treatment, they relapse.

DR. KING: What were the autopsy findings on the colts that died at two months?

DR. RILEY: We expected to find evidence of a rather severe colitis or enteritis, but were surprised to find that if they lived for many weeks and died from some other cause, the gut was absolutely normal. In a few of our cases, the liver was enlarged and cirrhotic but this is not a constant finding. Most cases do not die. If they did it would help us out.

DR. BURGER: I believe I have seen isolated cases of a similar condition and have attributed it to very heavily fed mares producing a great quantity of milk. I have had some luck by putting the mare on a very low feed ration. It may be a case of people killing the mare with kindness because these mares are very fat and milk like Jersey cows!

DR. TEMPLE: In California, we have encountered an unexplainable diarrhea which seems to be associated with nervousness. The horse looks good, runs well but has continuous diarrhea and does not put on quite the weight that he should. When treated

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with Entromycin or pectin-kaolin, the diarrhea slows down temporarily and the horse begins to put on weight. Treatment is effective for only a week or ten days, for as soon as the treatment is stopped the condition recurs. Does anybody have a treatment for this condition? I believe it is associated with nervousness while in training for a race.

DR. HILL: I have run into the same problem. Put a tablespoonful of lime in the drinking water and the diarrhea will stop.

DR. PHILLIPS: Have any members of the panel encountered isolated cases of chronic diarrhea in adult horses?

DR. BURCH: There are many and they pose many diagnostic problems. I would say that we see this condition most commonly in horses from the Argentine. There they apparently chop much of the green vegetation and when the horses are switched to a dry feeding program as is usually the case in this country, the change in management causes the diarrhea to persist for a long time. One thing I did not mention before is that the electrolyte solution should contain two to three times the normal content of potassium, because, regardless of the type of diarrhea there is a loss of potassium. In the acute condition, levels of 8 to 12 mEq of potassium should be used. The laboratory analysis will show how much additional potassium is required. One of the virtues in infusing amino acids is that when they start to come around, the prolonged recovery period can be shortened by multiple water-soluble vitamins and massive quantities of protein, both orally and intravenously.

DR. UMANS: With regard to the problem of improperly formed feces, not necessarily diarrhea, I have on some occasions used acidophilous milk with high potency vitamin B complex. It seems as if it helped some cases.

MODERATOR TEIGLAND: Incidentally with reference to chronic diarrhea, an article by Dr. Lundvall was published recently in the Journal of the A.V.M.A. concerning the isolation of an organism of yeast origin.

DR. WILLIAMS: We have had three cases like this, two terminating fatally before I could get to them and one which lingered for about three days. In the latter case, inclusion bodies were found in the liver. Can anyone here explain what these might mean?

DR. MILLER: Dr. Jensen and Dr. Adams came down from Fort Collins and took tissues from one animal that lived for about three days before dying. No inclusion bodies were found and on culture, only E. Coli was detected. In another unconnected outbreak, inclusion bodies were found in the brain. It was thought that this might have been some form of nitric poisoning.

PREGNANCY DIAGNOSIS IN THE MARE*

R. ZEMJANIS

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The growth of the horse population since World War II has reinstated the horse as a veterinary patient. The renewal of interest in horses has resulted in a more active horse breeding industry and also a rapidly increasing demand for veterinary services in the field of reproduction. To meet this demand more veterinarians are needed; more veterinarians competent in diagnostic methods among which pregnancy diagnosis is the foremost in importance. Regardless of the nature of the complaint and the type of service demanded by the client, any form of therapy must be preceded by pregnancy examination. It must be the first and basic step of the approach of any type of infertility problem. The disregard of this simple rule has very often led to embarrassing consequences.

Comparison of the two available methods of pregnancy diagnosis favors pregnancy examination by rectal palpation over laboratory tests in practically all aspects. The former method is not limited to certain periods of gestation as are the laboratory tests. It provides also more accurate and reliable results. The diagnosis can be relayed to the client without the delay necessitated by the forwarding of samples to the laboratory and the time required for the laboratory test. The fact that the rectal method provides reliable diagnosis at least twenty days earlier in the gestation period represents the most important advantage. An accurate diagnosis can be made as early as 30 to 35 days of pregnancy, thus providing ample time for treatment, if necessary.

There are clinicians who avoid rectal examination in pregnancy diagnosis. Some are influenced by the misconception held by certain clients that rectal examination is detrimental to pregnancy. Day[3] states that rectal examination for pregnancy does not appear to increase the incidence of prenatal deaths. Personal experience indicates that the 3 to 5 per cent loss of diagnosed early pregnancies is not higher than the corresponding loss in animals not examined before the fourth to fifth month following service. Such information, if tactfully presented to the client, is helpful in the elimination of misunderstanding.

There are also veterinarians who do not feel competent to employ this method. The competence however can be acquired readily by an effort to examine rectally all mares presented for physical examination, combined with a review of the theory of pregnancy examination. The purpose of this discussion is to present

*Approved for Publication as Miscellaneous Journal Series Paper No. 1070, Minnesota Agricultural Experiment Station, St. Paul, Minnesota.

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an outline of pregnancy diagnosis by rectal examination for such a review. It is clearly understood that the outline is not original, since the method is as old as the veterinary profession itself. These limitations are also valid for the excellent publications listed in the bibliography and meant for those interested in further study.

Pregnancy Diagnosis by Rectal Examination

This method is applicable to animals of all breeds except the smaller Shetland pony mares, the applicability depending upon the size of the animal and that of the examiner's arm and hand. Since the examination is objectionable to the animal, protection of the examiner and the animal deserves due concern and attention. It is beyond the scope of this presentation to list the numerous mechanical devices and methods of restraint. In selection of the means of restraint it is advisable to choose the mildest means possible considering the animal as an individual and the circumstances involved. Adequate protection can often be obtained from gates, partitions and hay or straw bales placed properly behind the animal. Likewise, mares might be examined while in the trainer or truck if the tailgates are low enough to permit safe examination.

Bandaging of the part of the tail in apposition to the perineal region and rectum prevents introduction into the rectum of hair that might traumatize the mucosa of the intestine. Soiling of the tail is also avoided.

The form and consistency of the fecal material in the rectum do not permit friction-free entrance into the gut. Proper lubrication facilitates introduction of the hand and is helpful in the examination itself.

Examination is also facilitated by fasting of the animal, if this can be arranged. Fasting diminishes the mass of intestinal contents and the genital tract can be more easily manipulated and examined.

Rectal examination being a method of internal examination requires a landmark or orientation. Such a landmark, having distinct characteristics and a relatively constant site in the mare is provided by the ovaries. Systematic examination for pregnancy is begun with the location of one of the ovaries. The left ovary is more easily accessible for a right-handed operator and vice versa. In the non-pregnant normal mare and animals in early pregnancy the ovaries are found in the sublumbar region 5 to 10 cm directly anterior to the upper portion of the iliac shaft. The ovaries are recognized by their distinct, oval and irregular outline. The ovaries measure from 4 to 7 cm in length and 3 to 6 cm in height and width. These dimensions vary with the size, the parity of the mare and the season of the year, the left ovary being the largest in practically all mares. The only structures that might possibly be mistaken for the ovaries are the formed fecal balls in the lesser

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colon. The ovaries are however easily distinguishable from the latter, which are softer in consistency, are numerous and are relatively freely movable. The extent to which the ovaries are movable is limited by the suspensory ligament and the uterus. Failure to locate the ovaries in the described position is highly indicative of advanced pregnancy, in which case the ovaries are found further anteriorly and ventrally as well as closer together. Detailed examination of the ovaries does not contribute information of significance for pregnancy diagnosis and therefore is omitted until the examination of the uterus is completed and the animal found to be non-pregnant.

After locating the ovary, the uterovarian ligament is followed medially until the abrupt, flattened ovarian end of the horn is detected. The uterus of the mare is almost T-shaped with the tips of the horns bent dorsally and anteriorly forming an arch with the lesser curvature facing dorsally. The body and the horns are flattened dorsoventrally. The anterior edge of the body, located on the pelvic brim, is 1 to 1½ cm thick, the thickness depending upon the size and the parity of the animal. The dorsoventral thickness of the horns tapers but slightly towards the ovarian end. The consistency of the organ is flaccid even in animals in estrus. Turgidity of the uterus is apparent only in early pregnancy and does not indicate the estrous state. In order to obtain reliable results, the entire anterior edge of the uterus and the horns must be palpated. Differences in size, shape and consistency between horns, within one horn and the body of the uterus are most easily detected by palpation between the thumb and the other fingers bent and slid underneath the anterior edge of the uterus. Palpation of the entire uterus in one single attempt might be impossible because of training and peristalsis. Examination should be resumed at the site last examined after this interference ceases. Occasionally the opposite horn might have to be examined starting from the corresponding ovary. Becoming thoroughly familiar with the characteristics of the non-pregnant normal uterus (Fig. 1) is extremely helpful, particularly for the beginner, in efficient pregnancy examination.

Pregnancy diagnosis is based upon detection of the physiological alterations of the uterus during pregnancy. The changes regarding the shape, size, consistency and position of the pregnant uterus are outlined in chronological order as follows:

a. Four week (28-30 day) pregnancy. This stage is characterized by a distinct spherical enlargement, measuring 2½ to 3 cm in diameter, located in the lower third of one horn, predominantly the right (Fig. 2). This is the time when the tonus of the uterus is greatest, permitting distinct palpation of the pregnant horn above and also below the bulge, which is bulging out primarily ventrally. Careful comparison of both horns and particularly the

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area of the bifurcation which protrudes in a downward curve in the non-pregnant mare as well, is imperative for detection of this stage of pregnancy.

b. Five week (35 day) pregnancy. The enlargement is still spherical in shape and still involves only the horn. The size of the distinct, circumscribed enlargement has reached 3 to 4 cm in diameter and resembles a golf ball in size and shape. The wall over the enlargement is thinning and permits the examiner to feel the fluctuation of the enlargement. The tonus of the uterine wall is relatively high.

c. Six week (42-45) pregnancy. The enlargement attains a slightly oval form measuring 5 to 6½ cm in length and approximately 5 cm in diameter (Fig. 3). The conceptus with its membranes and fluids has reached the junction of the horn and the body.

d. Seven week (48-50 day) pregnancy. The now distinctly oval enlargement is beginning to involve the body of the uterus. It is approximately 8 cm long and 6 cm in diameter. Fluctuation of the fetal fluids within the enlargement and fetal membrane are clearly palpable.

e. Two month pregnancy. Approximately one-half of the enlargement involves the uterine body. The enlargement now has the shape of a football and measures 12 - 15 cm in length and 8 - 10 cm in diameter (Fig. 4).

f. Three month pregnancy. The enlargement involves the entire body of the uterus and is approximately 20 to 25 cm long and about 10 - 15 cm in diameter (Fig. 5). The bulk of the uterus is abdominal and the ventral aspect of the uterine body cannot be reached. The conceptus can be easily detected upon ballottement of the enlargement.

g. The period three to five months of pregnancy. The taut anterior edge of the broad ligament directed downward, forward and medially is found in the site previously occupied by the ovaries. The now marked descent of the pregnant uterus exerts traction on the broad ligament resulting in markedly increased tension of the ligament. The ovaries are consequently found further ventrally, more anteriorly and also closer together. The horns of the uterus are found to be deflected medially over the enlarged body of the uterus. The descent of the uterus is not complete, permitting palpation of the fetus (Fig. 6).

h. The period five to seven months of pregnancy. The descent of the uterus continues and is completed towards the end of this period, resulting in still greater tension on the broad ligaments and further displacement of the ovaries. This displacement, the hypertrophied uter-ovarian artery and the removal of the ovaries and the uterus from the non-pregnant position are sufficient to establish a diagnosis of pregnancy.

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i. The period seven months of pregnancy to term. Ascent of the pregnant uterus begins at seven months of pregnancy. The fetus can be distinctly palpated.

The conditions requiring concern and consideration in differential diagnosis are relatively few and rare. The most important of these, pyometra, is easily distinguished from the pregnant status by the lack of a conceptus as well as the thicker wall of the uterus. In cases of doubt, vaginal examination might assist in making a diagnosis. Partial dilation of the uterus as described by Knudsen[5] might provide difficulties. Conditions corresponding to that description have not been reported elsewhere in the literature. It has not been encountered in this clinic and therefore cannot be commented upon. Mistaking the distended bladder or the pelvic flexure of the left colon for the pregnant uterus has been mentioned in differential diagnostic discussions[1, 2, 6]. Actually the most important and frequent cause of errors in pregnancy diagnosis by rectal palpation is the failure to ascertain that the organ or structure examined is the uterus. Even the experienced operator may encounter mares which do not cooperate and a definite diagnosis thus cannot be made . Failure to admit that diagnosis is impossible at that particular time is another potential and true cause of diagnostic errors.

The significance of vaginal examination has been pointed out by several workers[1, 2]. It is highly doubtful that positive findings obtained by vaginoscopic examination are adequate for a diagnosis "pregnant" in the absence of positive signs of pregnancy detected by rectal examination.

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DR. WISEMAN: Can you give us anything at all on tests? Do you have any information at all, other than palpation, on any laboratory tests of value?

DR. ZEMJANIS: You mean the test by which the presence of PMS can be detected or determined?

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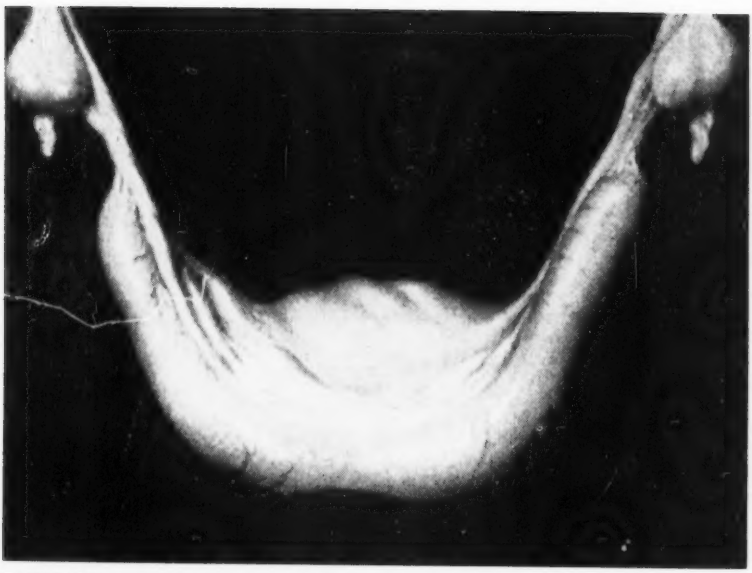
DR. WISEMAN: Yes.

DR. ZEMJANIS: Yes, we have the mouse test. Twenty to forty cc of blood is allowed to coagulate in the refrigerator and 0.25 cc of the serum is injected twice daily for two days into four or five mice approximately 3½ weeks old. The results are read 24-36 hours after the last injection by postmortem examination of the genital tract. Small, pale, pink ovaries and a hardly detectable uterus indicate absence of PMS in the blood of the animal tested. In the case of a positive test the size of the uterus is considerably increased, up to five times, and the organ is hyperemic. Depending upon the amount of PMS in the serum you will have superovulation and/or hemorrhagic follicles. This test is supposed to be reliable from 50 to 100 days of pregnancy. I would use it in Shetland mares that are too small for rectal examination and animals that are intractable. I do not think it is a reliable test since reliable results are obtained too late, namely 80 or 90 days after breeding. Where are you if the animal has been bred in May and has failed to show heat since? For the good of our clients they have to be sold on the early pregnancy diagnosis.

In this connection nobody has asked a question I have been expecting. I am advocating early pregnancy examinations. I am also sure that there are people in the audience thinking "What about the prenatal deaths following an early pregnancy diagnosis?" What about the cases where a mare has been declared pregnant and returns in heat two months or so later? Prenatal deaths present a definite hazard. I believe that we should attempt to explain this to our clients and make them understand the problem. Of course, whenever something suspicious is detected during pregnancy examination, the client should be informed and reexamination should be recommended.

In regard to tests for estrogens, we have used Cuboni's test, which is a chemical test. Any clinical laboratory can run it. Here again we have the problem of reliability. The positive tests are practically always correct, the negative, however, are not. This test is supposed to be reliable from 90 days of pregnancy. We have however obtained negative Cuboni tests in mares found to be pregnant by palpation.

We had an interesting case illustrating this. A gentleman farmer had bought two mares in May. In June I was asked to find out why they did not come in heat. I found one of them pregnant 100 days, the other 110 days pregnant. For student benefit we collected urine samples from these mares for Cuboni test. Since the owner was not present at the time the mares were examined I wrote a letter to him explaining that his two mares were pregnant. The Cuboni test gave negative results and the technician sent a report to the client without notifying me. I had to do some explaining to the poor client.



ZEMJANIS. Fig. 1. The genital organs of a normal, non-pregnant mare.

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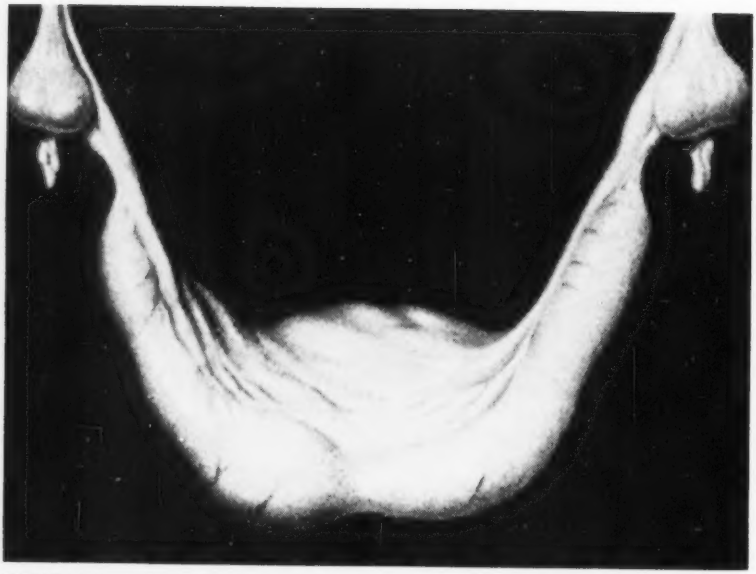

ZEMJANIS. Fig. 2. The genital organs of a mare, 28-30 days pregnant.

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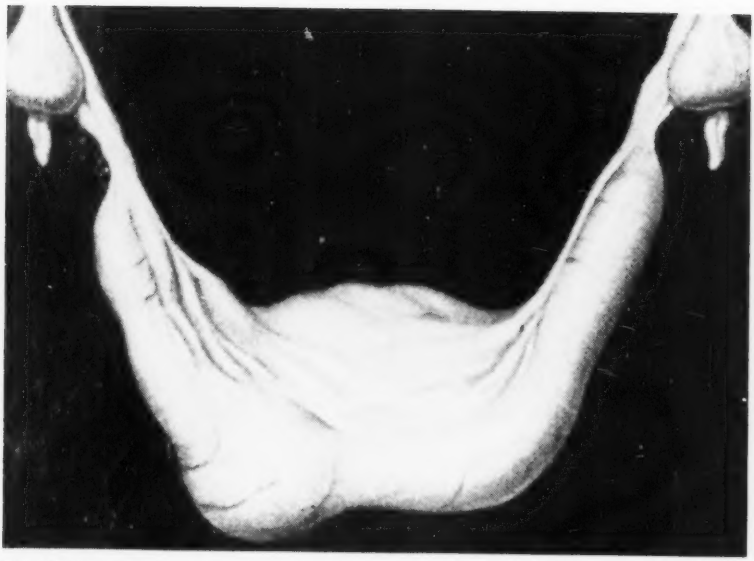
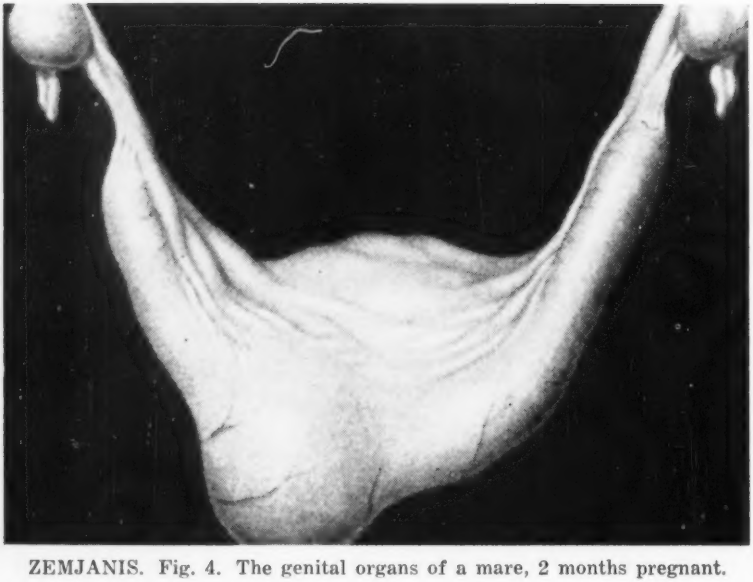


ZEMJANIS. Fig. 3. The genital organs of a mare, 42-45 days pregnant.



ZEMJANIS. Fig. 4. The genital organs of a mare, 2 months pregnant.

ZEMJANIS. Fig. 5. The genital tract of a mare, 3 months pregnant.

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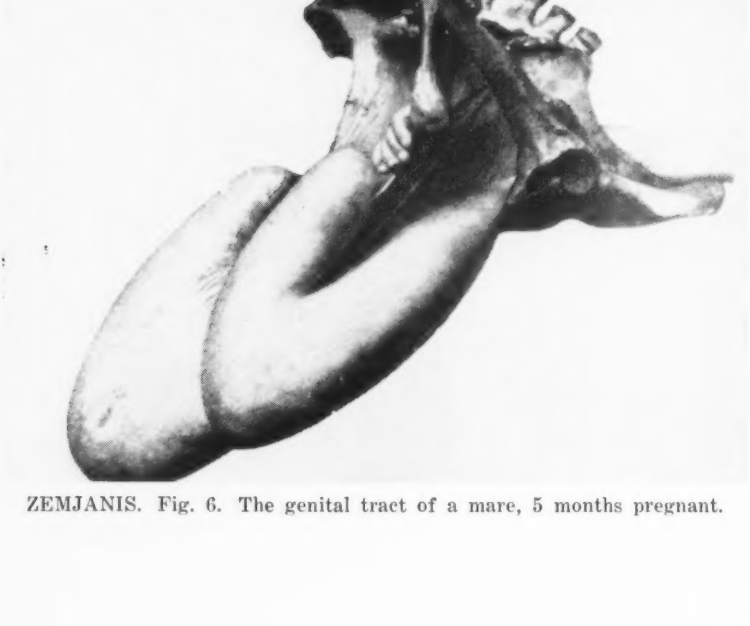

ZEMJANIS. Fig. 6. The genital tract of a mare, 5 months pregnant.

THE REPAIR OF PERINEAL LACERATIONS IN THE MARE

MURRAY E. FOWLER*

INTRODUCTION

Complete or third degree perineal lacerations in the mare present a difficult problem to the equine practitioner. Affected mares usually remain barren if correction is not made, and are also undesirable as saddle horses because of the wind sucking" associated with the flaccid, common opening of the rectum and vagina.

A technique first described by Goetze in 1929 may give the practitioner a simpler and more effective way of repairing these lacerations.

PRESURGICAL CARE

To repair perineal lacerations involving the rectum and vagina the rectum should be relieved of as much stress as possible by withholding hay and bedding for 4 to 6 days preceeding surgery. During this time the patient may be given bran mash and grain or ground alfalfa and molasses. For 24 hours preceeding surgery, all feed should be withheld.

Epidural anesthesia is used. If there are local areas of sensitivity these may be infiltrated to obtain suitable anesthesia.

After the epidural anesthesia has taken effect, the rectum is manually emptied as far forward as possible. A prepared ball of gauze and cotton is inserted to minimize contamination of the surgical field. A length of gauze should be attached to the ball and the outside portion tied to the tail.

Only rarely is it advisable to operate immediately after laceration only when the tear is associated with a minimum of trauma and there is little edema or necrosis of wound edges. Nearly always one should wait until the wound margins have healed and all edema and inflammation have subsided, usually in 3 to 6 weeks. These wounds heal quickly in spite of contamination. Peritonitis is rare, but is a possibility.

The mare's tail is bandaged and tied in an upward manner. (Fig. 1). The perineal area is cleaned and scrubbed with antiseptic

*Editor's Note. The reader is referred to "Repair of Perineal Lacerations in the Mare and Cow" by Otto C. Straub and M. E. Fowler in J.A.V.M.A. Vol. 138 (1936) p. 659.

†Nolvasan. Ft. Dodge Labs., Ft. Dodge, Iowa.

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soap. Tincture of Zephiran or 2% tincture of iodine is applied. The lacerated area is carefully cleaned with cotton which has been soaked with a mild antiseptic solution* until no traces of dirt are left on the cotton.

Since sterile technique is impossible in this area, a bucket is filled with a solution of non-irritating antiseptic* into which the hands or instruments can be dipped if necessary.

As the animal is being prepared for surgery, the surgeon must evaluate the laceration to determine the exact line of incision, and form a mental picture of the dissection of the rectal and vaginal mucosae to accomplish the repair.

INSTRUMENTS

A few special instruments are required, espescially when dealing with a long tear. Long needle holders and forceps are needed for working deeply. The suture material is Vetafil (0.4 mm).

SURGERY

The vulva is spread by placing a curved tonsil-seizing forceps on either side. An incision, parallel to and one quarter inch anterior to the muco-cutaneous junction of the vulva (Fig. 2), is started at the dorsal border of the vulvar mucosa and extended ventrally as far as appears necessary. Generally, the incision is made about one third of the height of the vulva in small lacerations, and almost one half in severe lacerations.

The mucosa, along with some submucosal tissue, is separated from the peri-vaginal tissue to make a flap extending from the vulva in a diagonal to the area where the rectum and vagina are clearly separated (Figs. 3 and 4). The same procedure is followed for both sides. Any scar tissue from unsuccessful repair attempts or from healing processes should be removed, for it hampers healing.

The ventral borders of the rectal mucosa are then freshened. Hemorrhage can usually be controlled with gauze sponges and pressure. The flaps on both sides should be as symetrical as possible. The existing shelf by gentle traction is pulled posteriorly (Fig. 5[A]). Undue tension should be avoided; the advantage of maximum closure is more than offset by excessive stress to the tissues, which inhibits healing. A long needle holder and a No. 00 half-circle needle are used to place the individual sutures (Fig. 5[B]) which, first, go through the vaginal mucosa deeply to include perivaginal tissue and then come out through this tissue to the region of the musculo-fascial defect. Next, the suture is placed deeply in the submucosal layer of the rectum on one side without penetrating into the lumen. The suture is then advanced to the other side and the submucosal layer of the rectum penetrated

there. Next, the suture is placed in the perivaginal tissue and then through the vaginal mucosa to the vaginal lume. The suturing technique is completed by passing the suture through both vaginal mucosal flaps back to the starting point where it is tied. It is of the utmost importance that the ties are pulled tight so that no slack remains, thereby, the rectal mucosa from both sides is pulled together as well as the vaginal mucosa. To facilitate removal, the individual sutures are numbered by tying knots into the ends of the individual sutures, starting with one, etc. These sutures should be cut 4 to 6 inches long so they can be grasped outside the vulva for removal, however, they should not be so long that they accumulate filth or tail hairs. Sutures should be placed ¾ of an inch apart. When all these sutures have been placed, the interior of the rectum should be carefully examined for any suture that can be palpated. Any such suture should be removed and need not be placed if the other sutures are all satisfactory. The rectal pack is removed at that time.

With the new shelf completed, care should be taken to furnish a good connection between the shelf and the sphincter ani. This is accomplished with single mattress sutures placed into the sphincter muscle and deeply in the upper portion of the vulva, thereby, avoiding a possible pocket directly in front of the sphincter. The upper part of the vulva is then sutured, either by simple interrupted sutures or by vertical mattress sutures.

ARTERCARE

Following surgery the animal should be given no hay. Ground alfalfa and molasses or bran mash and grain are satisfactory. This diet should be fed until the sutures are removed, in 10 days or 2 weeks. Antibiotics such as penicillin and streptomycin should be given for 5 to 7 days. Occasionally the vulva is too small for natural breeding following surgery. However, the patient can be artificially bred or the vulva opened by episiotomy. Episiotomy may become necessary prior to parturition. The owner should be advised to call a veterinarian at that time.

SUMMARY

This technique closes the rectal mucosae as if a subcuticlar suture were used. Secretion of serum seals the rectal wound shortly after surgery.

This method is adapted to any perineal laceration and provides a thick, strong shelf between the rectum and the vagina with a minimum of suturing. No sutures penetrate the rectal mucosa, so that infection is minimized.

No packing is necessary between the two mucosal layers, since all this space is closed with sutures. First-intention healing is

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anticipated. This technique is best suited to third-degree perineal lacerations, but is also adaptable to the repair of recto-vaginal fistulae.

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DR. WISEMAN: How long is the needle holder?

DR. FOWLER: About twelve and one-half inches.

QUESTION: How far apart are the cutures placed? Half an inch?

DR. FOWLER: They would have to be a little more than that; perhaps three quarters to one inch apart.

QUESTION: What about the problem of foaling?

DR. FOWLER: I think I may have given the impression that the shelf is a couple of inches thick. Actually this is not the case. When the tissues stretch and return to normal, the shelf is not much thicker than it was prior to injury. As a rule, little trouble is experienced at a future foaling. I think that the owner should be warned about this possibility, however, and an examination made to determine if difficulty is likely to be encountered.

DR. GUARD: I would like to say a word in support of this technique. I like the description of it although I have not used this method. I use an entirely different technique, one much more difficult than this! I have had plenty of failures too! In those cases where a foot is forced into the rectum and withdrawn, the hole can be repaired with a purse string suture immediately after injury. It does not matter if fecal contamination is present or not. These animals never miss a feed; the wound heals rapidly. I think it is better not to wait till scar tissue forms but if there is much tissue reaction due to injury then a waiting policy should be adopted.

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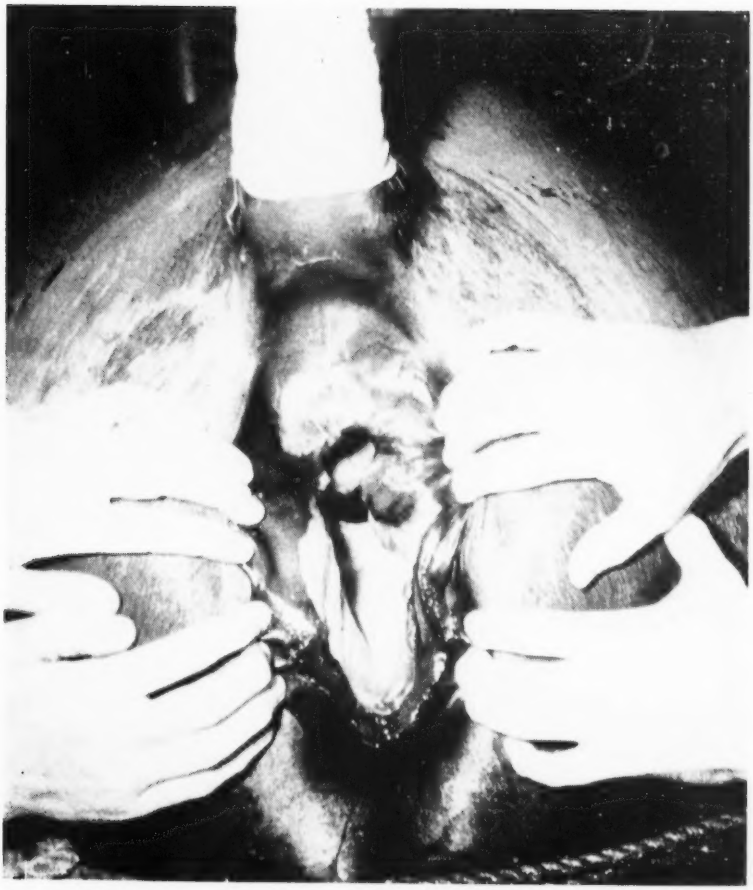
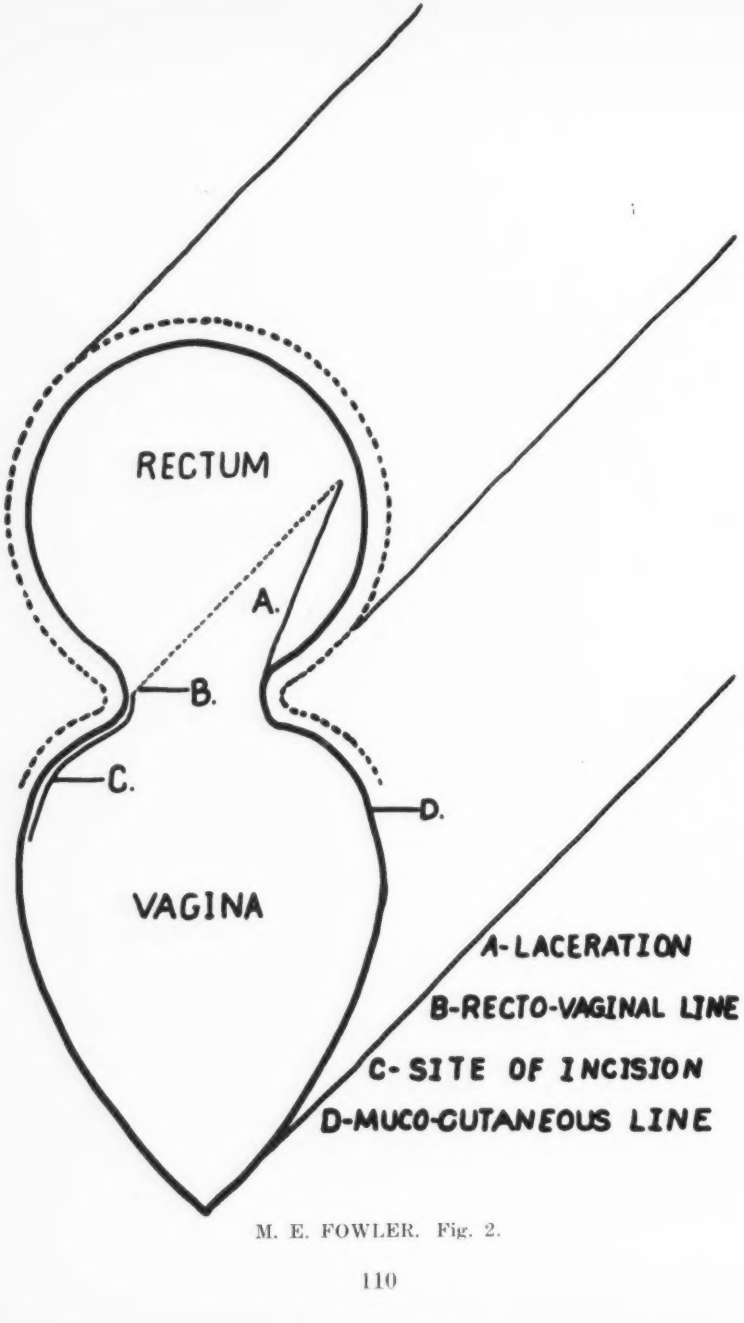
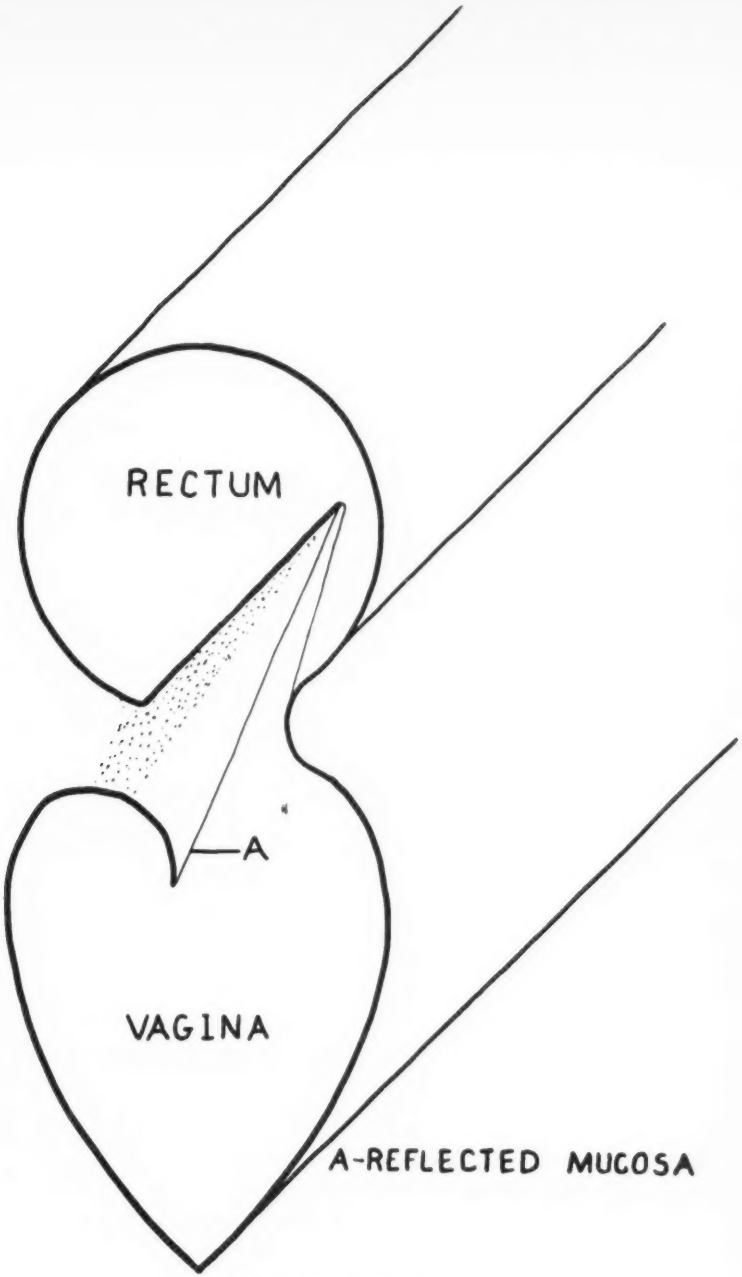
M. E. FOWLER. Fig. 1.



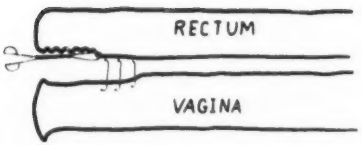


M. E. FOWLER. Fig. 2.



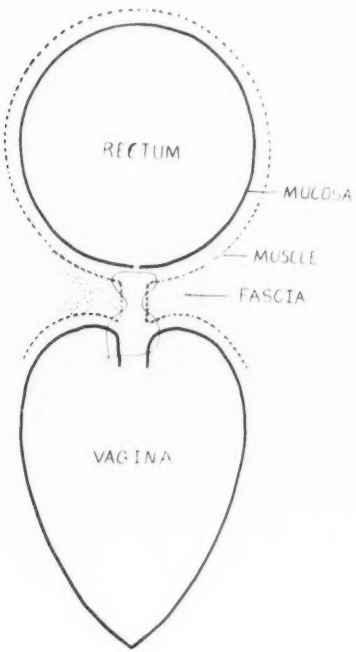
M. E. FOWLER. Fig. 3.

M. E. FOWLER. Fig. 4.



A-PULLING SHELF BACK

M. E. FOWLER. Fig. 5 (A and B).



SUTURE PATTERN

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AFRICAN HORSE SICKNESS

by

DONALD MILLER

My mission here today is mainly to alert you equine practitioners to the seriousness of the threat of African horse sickness.

A serious outbreak of African horse sickness has occurred outside an area which, until this year, was thought to be the natural domain of the disease. A prominent veterinary official of the foreign agricultural field has commented that it is one of the classical outbreaks worthy of the intensive interest of epizootiologists throughout the world, yet veterinarians in this country have remained aloof.

The disease by spreading through countries of Asia, Africa and the Mediterranean area, now poses a very real threat to the horse population of the Americas.

We can no longer remain aloof to this threat, and protective measures should be, and are being established, to protect the horse industry of this country. Perhaps these protective measures are late; we hope not too late.

African horse sickness is an acute or subacute febrile, infectious, viral disease of the horse species.

The disease has recently spread from its former confined epizootic areas of Africa, to the Middle East and Asia. It has been identified in Cyprus, and now threatens the European Mediterranean area and possibly, all areas of the world.

The disease invaded areas of Iran and Pakistan in the fall of 1959 and was quiescent during the winter months; then in the spring of 1960, spread eastward to India and westward to Turkey and later, Cyprus.

The magnitude of the losses sustained is expressed in the following report:—

In Iraq, twenty thousand Arabian horses died, and hundreds more were dying daily. In Turkey, 10,000 mules and horses died in addition to 5,000 which were sacrificed in an effort to stop the spread.

This has been an epizootic rivaled in invasiveness and rapidity of spread in recent years only by the great Asian influenza pandemic of the late 1950's.

The morbidity and mortality of African horse sickness are spectacular in horses and mules. As many as 90 per cent of susceptible horses may die. Donkeys are less susceptible but have harbored the virus and contributed to the spread of the infection. In addition to the equine, the disease infects dogs which eat the meat of infected horses, and it may be serial-passaged in dogs. Goats are susceptible by inoculation, but the virus cannot be serial-passaged in goats.

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The ferret is susceptible and is used in isolation studies. White mice and guinea pigs are also susceptible and rabbits are used as laboratory animals, for the preparation of antisera.

The economic aspects of the condition are great in countries where equine draft and transport power is involved. This is especially noted during harvest and planting seasons.

The causative agent of African horse sickness is a viscerotrophic virus transmitted by the Culicoides species of gnat, which is widespread in this country and this is also the insect which transmits blue tongue of sheep. Other insect species have been incriminated but they are less likely vectors. The Culicoides is a blood-sucking insect, and deposits its eggs in water, or wet areas.

African horse sickness is not transmitted by direct contact. Equines may harbor the virus up to ninety days, but the reservoir during the periods the disease does not occur is not known. This is a very serious matter, requiring research, as it is extremely important to know the reservoirs of the virus, if we are to prevent its entry into this country.

There are four separate clinical forms of the disease. These are the febrile, pulmonary, cardiac and mixed forms. There appears to be little consistency in the manifestation of specific forms, except in the febrile form which occurs in horses where previous immunity has broken down, or in less susceptible equine species.

The temperature may be 105° F. after three days in the febrile form, or 106° or more in the pulmonary and acute forms. In virulent outbreaks, the pulmonary form is most common. There are all the manifestations of acute pulmonary edema. A voluminous discharge of yellowish liquid or froth from the nostrils may be noted, especially after paroxysms of coughing.

In the uncomplicated cardiac form, there may be extensive swellings of the head and neck regions. The lesions in the pulmonary form are lung edema and hydrothorax. There is a yellowish, gelatinous infiltration of the subcutaneous, subserous and intramuscular tissues of the head, neck and thorax.

Hydropericardium is a constant lesion, and there are diffuse hemorrhagic lesions in all the cardiac tissue. Diagnosis is accomplished by symptoms, pathology and epidemiological considerations. In certain cases, anthrax and clostridial infections may be confused.

There is available a complement fixation test which is not type-specific, and also an intracerebral virus neutralization test in mice where rabbit antisera is used. To complicate this picture, there are 42 known strains of the virus, with seven immunogenic groups. The virus is reported as naturally viscerotrophic but may be neurotrophically adapted by serial intracranial passage in mouse brains.

Alexander of South Africa, has propagated the neurotrophic

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virus in developing chick embryos. The virus remains visable only for short periods, under natural conditions, but may be maintained in desiccated brain material for a very long period.

With regard to immunity, naturally infected survivors are solidly immune to homologous viral studies, but the multiplicity of strains naturally requires a polyvalent immunization.

A polyvalent intracerebral mouse-brain passage vaccine is effective. A virus must be passaged at 100 intracerebral mouse brain levels, and twelve adult mouse brains are required to produce 600 doses of the vaccine. Virus-serum immunization or inactivated tissue has been found of little value.

It is believed that the vaccine will protect up to six years but breaks of up to 15 per cent have occurred, possibly due to a highly invasive virus of a particular strain. Ordinarily, breaks involve no more than 1 to 2 per cent of the animals vaccinated.

What is being done to protect the horse population of this country and to alleviate the condition in the affected areas?

In July, 1960, the Federal Animal Inspection and Guarantine Division ordered a guarantine of at least thirty days of horses coming from Africa, Asia and certain Mediterranean countries. This quarantine order requires that the horses be held in insect-proof quarters, and the use of No. 40 mesh since the Culicoides gnat is a very tiny insect.

The animals and their quarters on the transporting plane or ship are treated with an insecticide aerosol of pyrethrins and DDT, before unloading. Temperatures are taken regularly of horses in quarantine and, in suspicious cases, blood is inoculated intracerebrally in mice to detect any virus and, if necessary, virus neutralization tests would then be conducted.

Inoculation of susceptible horses has also been carried out.

Under United Nations auspices, veterinary officers in the Middle Eastern countries are being trained in Iran and Pakistan in diagnostic procedures and vaccine production. Vaccine production is being pushed in countries able to do this work, such as South Africa, Kenya, Egypt, Pakistan and Iran.

Recently, representatives of the Department of Defense, the State Department and the Department of Agriculture have met and formulated suggested programs for the control of the disease in Africa and Asia, with the view of preventing its spread to Europe and this continent. It is believed that such a program would require a half million doses of vaccine a month for three months starting in April, 1961. The two countries having the capability of producing vaccine, Iran and Turkey, are able to produce about 167,000 doses of a vaccine a month.

The agencies mentioned have outlined procedures for the production and administration of vaccine, and the diagnosis of the disease but, the problem is a major one under conditions existing in the area.

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In this country we must be prepared to prevent the entry of the disease, to diagnose it promptly, should it enter, and eradicate it before it gains a foothold.

To gain more knowledge of the disease, the production of vaccine and diagnostic measures, we have assigned one of our men from Plum Island Research Laboratory to South Africa. This is a routine procedure. In recent years we have had veterinarians from our research and regulatory offices working in foreign countries where these diseases exist, so that we will be better able to prevent their entrance and, also, to more assuredly eradicate them should they get in here.

In regard to preventing the entry of the disease into the United States, I wish to point out a weakness in our animal quarantine laws which govern the importation of animals from foreign countries. Presently, these laws do not provide authority to prohibit the entry of certain classification of animals, when such action is necessary to protect the livestock industry of this country. This is the case with horses. We may merely place certain regulatory restrictions on horses entering from infected zones, but we may not actually prohibit their entry under the present laws.

Senate Bill 864 (860 in current session) which has received support from a number of livestock organizations provides, among other things, added authority for further protection against the dissemination of diseases of livestock and poultry.

In view of the African horse sickness situation, I believe this Senate bill merits your consideration and support.

If there are any points you wish clarified, especially from the standpoint of regulatory activities, I will be glad to attempt to answer your questions.

DR. COSGROVE: How long does the fly carry?

DR. MILLER: The fly, as far as I know, can remain a carrier for at least a month. This is a disease which tails off after frost, but apparently the reservoirs have been established in these newer areas of Asia and Africa and in Turkey and Cyprus. So, it is a disease that is recurring each year.

DR. GUARD: Have any human beings ever been affected by it?

DR. MILLER: I don't know. The question hasn't come up in any of our discussions.

DR. GADD: You said they do not pass by direct contact?

DR. MILLER: Yes, sir.

DR. GADD: How do they get the disease?

DR. MILLER: The Culicoides gnat will transmit the disease from animal to animal. This gnat will pick up the disease from the reservoir, whatever it is, then pass it to the horse, and then transmit it from horse to horse.

RESPIRATORY PROBLEMS OF THE HORSE; THEIR DIAGNOSIS AND TREATMENT

Panel: H. W. Johnson
E. A. Churchill
D. D. Delahanty
M. E. Fowler

DR. CHURCHILL: Rather than discuss several infectious diseases and their relation to the respiratory system, our panel members have selected a few topics for discussion. The first speaker, Dr. Harry W. Johnson, will talk about the chronic cough. This paper is based on work carried out by Dr. Johnson and Dr. G. M. Beeman.

DR. JOHNSON: My purpose on this panel is to discuss the use of certain products and what may be expected from the use of these products in the treatment of different types of respiratory disturbances in the horse.

I like to regard the non-specific equine respiratory conditions, such as the purulent nasal discharge with or without fever or cough in the same way that one regards these conditions in the human. After a man or horse has passed through the acute phases and recovery is progressing, we find in many instances that the sequel is in both species, a cough.

My interest in this respiratory syndrome in the horse extends over many years but the conclusion has been reached that a cough in the absences of fever is probably the complication which causes the greatest concern to horsemen.

The similarity of the syndrome in man and horses prompted the thought that perhaps some drug or agent used in man could also be employed with beneficial results in the horse. The first of these was a Sharp & Dohme product — Pertussis 'Serobacterin" Vaccine Mixed — a standardized suspension of chemically killed bacteria in P.S.S., "sensitized" by hyperimmune rabbit serum. The bacterial content is comprised of Hemophilus influenzae, H. pertussis, Micrococcus pyogenes, Staph. aureus, Staph. albus, Strep. hemolyticus (Group A, types 1, 3, 5, 6, 12, 17 and 19), Diplococcus pneumoniae (types 1 through 8 and 14) and finally Micrococcus catarrhalis.

The question arises — "Do these organisms cause infection in horses?" The answer is unknown but it is possible that some relationship exists. We used this vaccine on about 400 horses, after starting from scratch and using it on practically every type of infection which we encountered. It was decided finally, after a

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period of trial and error, that the vaccine was likely to produce best results in the horse suffering from an afebrile cough. The dose was 4 cc injected subcutaneously on the left side of the neck. It was noted that for beneficial response, a reaction at the site of injection was necessary. In other words, if no reaction occurred the cough was unaffected. The local reaction which may occur in a minimum for four hours varies considerably in size. The systemic reactions also varied, some being so severe that, 12-18 hours after injection, the horse was so stiff that it could scarcely move about the stall. Despite the obvious objections to systemic reactions we noted that the more severe the reaction the more readily would the horse recover. Recovery from a severe reaction took up to 72 hours with disappearance of the cough. The horse could then be returned to work.

If no reaction occurred, the horse was re-injected, usually 48 hours later. Although success did not follow every treatment, there was sufficient benefit to justify the use of this line of treatment.

With a few exceptions, it was of little or no value in other conditions. In a few cases of the less frequently encountered severe febrile cough, the results were excellent, the fever abating in 24 hours and the cough disappearing at 48-72 hours.

Another human product — catarrhalis serobacterin vaccine mixed — was used during a twelve month period on 87 horses up to two years of age. Again it was evident that a lack of reaction at the injection site meant a poor final result. Even in those cases which showed an evident reaction about the only benefit obtained was a reduction in severity of the respiratory condition. The cough was not controlled.

Unlike the pertussis vaccine, the catarrhalis bacterin does not contain Hemophilus influenzae; the assumption could then be made that the influence of the latter organism was the reason for our favorable results. Pertussis vaccine containing only H. pertussis was of no value in our work.

With regard to Mixed Bacterin No. 1, our experience shows that it is of little value in the immunization of horses against upper respiratory infections.

In an attempt to create an immunity against these conditions, we resorted to the use of an autogenous bacterin and most favorable results were obtained from a vaccine containing beta Hemolytic Streptococcus and Pseudomonas. Apparently in our locality these two types of bacteria play an important role in upper respiratory infections. Seventy-five horses have been vaccinated and preliminary results are encouraging. Where the disease has occurred despite vaccination, we believe that the severity of the condition has been decreased. An initial intramuscular injection of 3 cc is followed in young colts by a second dose of 3 cc 5-7 days later, the time of the second injection depending on the reaction to the

first inoculation. In older horses, a second dose of 5 cc is given, followed if necessary, by a third dose of 5 cc. The intramuscular route is preferred since subcutaneous injection prompted too great a reaction.

Usually within 24 hours of injection, there is a reactionary rise in temperature of 1-2 degrees. This frequently subsides and the temperature is back to normal by 72 hours.

This vaccine has also been used therapeutically along with antibiotics and sulfas. In many cases it seemed to hasten recovery and it appears that less relapses follow its use.

From the investigations reported, it is thought that a pertussis vaccine in combination with those organisms commonly present in upper respiratory infections in the horse might well lead to a more effective method of control for this syndrome. The assistance of the trained personnel and facilities of a commercial concern are necessary for the production of a possibly satisfactory method of immunization.

DR. CHURCHILL: To stay on a more or less practical basis, I will discuss a problem common to all equine practitioners, especially those who work at the race track. I have termed this problem "inspiratory dyspnea". Actually, this condition is not a disease but a symptom of so many conditions involving the interrelated respiratory upsets of the horse, that consideration should be given to the diagnosis or differential diagnosis of these conditions in which it is a cardinal symptom.

Mechanical Obstructions:

First thoughts are directed to the tongue of the horse. I believe that tongue swallowing, to the extent that it interferes with breathing, is sometimes the result of rather than the cause of inspiratory dyspnea. However, many horses, particularly those that tend to be nervous or those that are pretty much "on the iron", as they say, have a tendency to retract the tongue especially when ridden at high speed. When one takes hold of such a horse, the tongue will frequently flip back and snorting or wheezing results. Fortunately, most of these cases can be corrected by means of a tongue strap.

Occasionally, one meets the horse which defeats all efforts to prevent tongue retraction and the noise continues no matter how the tongue is tied. When this occurs, it pays to look for another cause for the noise, for as mentioned already, this could be the result of inspiratory dyspnea rather than the cause.

Neoplasms of the nasal passage are fortunately rather uncommon. In most cases, the condition is unilateral and involves polyp formation somewhere along the nasal cavity. Diagnosis is not difficult. There is usually a considerable difference in the volume of air entering and leaving each nostril, which can readily be determined by placing the hands in front of the nostrils. In

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some cases the polyps can be seen directly with the aid of a light or indirectly with a laryngoscope.

Neoplasms may involve the sinuses but this is seen rather infrequently considering the incidence of tumors in the horse. Usually they are classified as epidermoid carcinoma, myxoma or osteoma.

Another relatively rare but troublesome condition responsible for inspiratory dyspnea is elongation of the soft palate. This elongation or ventral extension overlaps the epiglottis and causes an expiratory noise as well as inspiratory dyspnea. This is sometimes of value in making a diagnosis. Eventually, the palate may become thickened, edematous and commonly infected due to trauma from the constant flutter. Occasionally, one sees a slight hemorrhage from the nostrils. The thicker the palate, the greater will be the trouble till finally a dead end is reached.

The carriage of the head or neck may be a cause of mechanical obstruction. This is more of a problem with gaited or saddle horses where a high carriage is considered desirable. Occasionally this causes such an oblique track through the pharynx that the latter will be occluded sufficiently to cause an inspiratory dyspnea. It is difficult to do much for this condition if this narrowing is due strictly to a matter of mechanics and not to any thickening of the pharynx. Some horses are naturally thick or heavy in the throat and when they show dyspnea on account of the head carriage there is very little that can be done about it. If, on the other hand, the head carriage is influenced to some degree by thickening of the pharynx, the prognosis is not so grave.

Bacterial Infections

Under this heading may be placed what I refer to as a plain, common cold, where the nasal mucous membrane thickens, becomes slightly edematous and partially occludes the air passage. This causes something of a flutter on inspiration but usually does not persist long enough to be of any consequence.

Sinusitis, by extension of inflammation to the nasal cavity, can cause edema of the nasal mucosa resulting in inspiratory dyspnea. Chronic sinusitis, in which the cavities are filled with pus causes a narrowing of the nasal passage with consequent inspiratory dyspnea. Pharyngitis is a major consideration. My estimate is that more than 50 per cent of cases of inspiratory dyspnea are caused by pharyngitis or rather, chronic pharyngitis because, when a horse has an acute condition or acute pharyngitis, it is recognizable and is expected to cause respiratory difficulty. When the condition does not clear up the pharynx becomes thickened, the dyspnea will persist over a long period and is frequently difficult to clear up.

The pharynx is very common reservoir of chronic respiratory infection. Many of you will recall Dr. Teigland's paper on this

subject when he discussed the culturing of the pharynx and the various infections involved.

Laryngitis may be an extension of infection from the upper respiratory tract and since the larnyx of the horse is relatively narrow, any swelling or edema will occlude the lumen with inspiratory dyspnea as a result.

Laryngeal Paralysis

The third heading is paralysis or paresis involving the larynx, hemiplegia, paralytic laryngitis or however one wishes to term the condition. The term "roarer" is familiar to all.

With regard to differential diagnosis of the conditions already mentioned, I do not feel qualified to make a diagnosis by standing out in the middle of a race track and have somebody breeze a horse or drive him in a cart while I listen to the horse as he passes or is pulled up abruptly in front of me. They sound too much alike to me and I have been fooled too often. I might take a guess as to what the trouble is but would not express my opinion until a further examination has been made. About the only time I like to see the horse actually work is when I wish to satisfy myself that the trainer or driver knows what he is talking about, when he says that the condition is inspiratory and not expiratory in character. Some people are just not able to differentiate between the two.

By starting the examination in the stall and questioning the men concerned, one can get some idea of what is going on. For instance, I try to find out whether the horse makes this noise constantly or only at top speed. Does it develop after he gets past the half-mile post? When does it start to appear? Does it become increasingly worse? Does the horse shown any sign of distress as a result of the dyspnea? Does he appear to weaken at the end of the distance for which he is being trained or raced? Does he wobble? When pulled up, what does the horse do? Is he exhausted? In other words, when pulled up, is he actually out of breath or does he give just one gasp and then breathe perfectly normally and not blow any harder than other horses accompanying him over the same route?

A standardbred, experiencing trouble with tongue swallowing or head carriage will make more noise when behind the gate, getting ready for the start, than he will during the race, because he is being held in. This, plus his eagerness to "run over the gate" aggravates the condition. When the driver "lets the horse have its head" so to speak, the noise will not be apparent. This indicates that "taking hold of the horse" has something to do with the production of the noise, therefore the tongue or head carriage is probably the cause.

It may be necessary, to examine the horse on more than one occasion before reaching a conclusion. Laryngoscopic examination is almost a necessity because it short-cuts so many of these

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examinations and gives much information that would not otherwise be obtained. The laryngoscope is not difficult to use. When introduced up the nostril, by rotating the tube one can observe the condition of the nasal mucosa and the openings into the maxillary sinus. Sometimes on the nasal mucous membrane one sees necrotic areas or erosions due to chronic infection. When the end of the laryngoscope has reached the cavity of the pharynx, one can see the roof and side walls of the pharynx and the soft palate lying below. Ventrally and further forward lies the opening of the larynx. The pharyngeal mucosa is smooth and in color resembles that of the mouth. All degrees of infection may be present in pharyngitis. Sometimes the pharynx appears reddened while on other occasions it may be both red and thickened, resembling corrugated cardboard, in that it lies more or less in folds. One may notice areas that appear white, the coloration being due to the formation of an overlying large plaque of mucus. On other occasions, this white color is due to erosion and necrosis of the mucosa. Just beyond the entrance of the larynx, the vocal cords may be seen and a decision made as to whether there exists paralysis or paresis involving the cords. For many years this has been a standard procedure in the diagnosis of a "roarer" and we considered it an almost foolproof diagnostic method. If, for example, the vocal cords were straight and tense, the horse was not a "roarer", but if one vocal cord sagged (usually the left one) the horse was a "roarer". However, it is not quite as simple as that. I have opened the larynx of horses which apparently had normal vocal cords. This was done because nothing was found on clinical examination and there might have been something abnormal within the larynx. I found that, although the cords appeared to be straight, they had very little tension and, as far as I am concerned, could easily have caused a flutter. On the other hand, I have commenced operating with the intention of removing the saccule only to find that the ventricule itself had a great amount of tension, which should not be present if paralysis exists.

At the moment, I am just a little confused and do not know how to explain it except to say that I am not sure that one can diagnose a "roarer" every time by means of the laryngoscope.

The surgical procedure which I use to correct the condition might be called the University of Pennsylvania method, because it has been taught there for so long as opposed to the method where a burr is employed which method is more commonly taught in other schools.

Instruments required are a scalpel, nicking knife, curved bistoury, straight Rochester-Pean forceps, curved Rochester-Pean forceps and occasionally a pair of 7″ tonsillectomy scissors.

The landmark for the operation is determined by palpation of the area on the upper, anterior aspect of the neck. By running the fingers up the rings of the trachea, one finally comes to a seem-

ingly enlarged tracheal ring with a prominence on the bottom — the cricoid cartilage. About three inches anteriorly will be felt another prominence, the body of the thyroid cartilage. The site of incision is between these two prominences. The area is prepared for surgery.

About 25 cc of 2 per cent procaine is used to anesthetize the skin and underlying tissues. The incision is made directly through the mid-line between the muscles so that hemorrhage will be minimized. The crico-thyroid ligament is then incised in the mid-line, the incision extending only as far as the thyroid cartilage anteriorly and the cricoid cartilage posteriorly. This permits maximum exposure of the interior of the larynx. Formerly, some operators cut through the cricoid cartilage but poor healing resulted with consequent increased occulsion of the larynx, so the method was abandoned.

On entering the larynx and before anesthetizing the interior, I examine the ventricules. This is a most important part of the whole procedure. When I insert my finger — I alternate fingers, depending on which side of the horse I am standing — into the ventricule, I try to estimate the amount of tension on the margin of the ventricule. In my opinion, one can determine the prognosis entirely on the basis of this tension. The more tension there is, the less successful will be the operation, if completed. I believe this strongly enough that I refuse to complete the operation on any horse in which the tension is sufficiently great that the operator has to work at getting his finger through the ventricule into the saccule. I do not think this is entirely without reason, for if the larynx is in any way paralyzed, the ventricule will be more or less gaping and flabby. It stands to reason that if one removes a ventricule or saccule when there is sufficient tension to obstruct the finger, there is going to be enough tension to prevent the formation of an adhesion firm enough to overcome the flaccidity. This type of case actually falls into the non-"roarer" category and I believe that surgery on such cases is responsible for giving the operation such a bad name among certain horsemen. In other words the high failure rate in this operation is probably due to our operating on the wrong horse and as a result, some horsemen are convicted that surgery is not indicated under any circumstances. On the other hand, if the ventricule is sufficiently flabby, one can count on the development of sufficient adhesion to tighten up the vocal cord and arytenoid.

When continuance of surgery is indicated the ventricule and interior of the larynx is sprayed by means of a DeVilbiss atomizer with 2 per cent butyn sulfate. After waiting a few minutes for the anesthetic to take effect, the index finger is inserted into the saccule, and guided by that finger, the medial margin of the ventricule is incised by the nicking knife. In other words, in a left sided paralysis, I would be standing on the right side of the horse

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and would have my left finger in the left ventricule. The incision would thus be made across the back of my finger, so to speak, or on the medial margin of the ventricule.

This incision should be long enough to permit the introduction of the finger without having to do much tearing with the finger nail. The horse tends to resent this part of the operation but if one is unhurried, it can be done efficiently. The finger is placed in the incision and directed towards the base of the saccule, the depth of which was ascertained during the examination for the degree of ventricular tension. This should not be carried out in a hurry. Experience shows that it becomes more simple with subsequent operations. Since the finger is still external to the saccule, the latter by flexion of the digit can be everted completely in a large thoroughbred, hunter or horse of similar size. In smaller horses, such as some standardbreds or saddle horses, there is not quite enough room to do this. In these horses, I loosen the mucous membrane as much as I can with the finger and insert the straight pair of Rochester-Pean forceps through the ventricule, grasp the bottom of the saccule and start to pull it inside out. The curved forceps are now applied to the membrane and by turning the forceps handle, the mucosa is rolled up on the forceps at the same time the finger strips the edges until the entire saccule is everted and on the forceps when it can either be snipped off with the tonsillectomy scissors or cut off with the curved blunt bistoury.

I have no fault to find with the burr method except that in some cases, chronic laryngitis has developed due to imperfect removal of the saccule by the burr. I have operated on a number of horses in which the burr had been used and the saccule not everted the result of which was nothing more than a scarification of the saccule by the burr. I believe that one can remove more mucous membrane by dissection than by the burr and that the former method implies a cleaner operation and a more certain result.

DR. DELAHANTY: In 1845, Gunther of Hannover, Germany, gave a comprehensive report of his findings with the use of different technics, which consisted of the removal of the ventricule, the removal of the vocal cords alone and arytenoidectomy. His original work was set aside and largely ignored. Early surgical correction was a major effort under general anesthesia but over the years the technic has been simplified to a standing procedure with a minimal incision such as Dr. Churchill described. We do not feel we have reached the end-point for we are not entirely pleased with our results, but mention will be made of this later.

The differential diagnosis is quite a problem, however, we will mention symptoms or findings, all or some of which it is desirable to observe after bacterial and mechanical factors have been eliminated.

Among the primary causes such as atrophy, i.e., the neurogenic failure of certain laryngeal muscles to perform their work, the primary one was indirectly touched upon by Dr. Churchill when he mentioned the tension around the ventricular orifice. The tension is caused by the thyro-arytenoideus muscle. It is in the form of a double band, one band lying anterior to the ventricule and the other forming the vocal cord mass. The second muscle is the cricoarytenoideus dorsalis which tends to elevate and pull to the side the arytenoid cartilage and thus allow phonation and increase in lumen diameter of the larynx during inspiration. This is a key muscle and is one of the group of muscles supplied by the recurrent laryngeal nerve.

A basic question is why do these muscles become involved? There have been the classical references to post-upper-respiratory-infection nerve damage, lead poisoning, chick peas, the hunter that stretches his neck, the horse in dorsal recumbency during surgery with the head extended and the nerves stretched. Polyps about the base of the larynx and aortic pulsation during strenuous exercise leading to nerve degeneration have also been considered. It must be admitted, however, that the actual cause remains unknown.

A desirable factor in the case is a history of a previous upper respiratory infection. This is the horse which shows a cough, runny eyes, a febrile reaction and is off work and out of condition for one to two weeks.

A history of an impatient trainer is appreciated. He goes ahead and works the animal quickly after cessation of nasal discharge and the horse looks healthy. Perhaps the horse is not really healthy, there may be a residual laryngitis or other complications. There may also be a history of respiratory infection in stablemates and if some of these animals are worked in the prodromal stage, nerve degeneration can ensue. This is probably our most sought-after point in the case history.

The great problem concerning the sound made by a "roarer" is the ability to recognize that it is made on inspiration.

With most of these "roarers" there is an increase in noise when there is an increase in stress and degree of work — no matter whether one is dealing with an old draft horse or a race horse of any type.

With regard to the ancient test for grunting, if anybody struck me twice with a cane, I think I would catch my breath and wait for the third blow! It is used and occasionally may have value. It is good, probably because some horses give a rather prolonged groan along with the grunt.

Some of our older textbooks, namely those by Law, Percival and O'Connor, refer to a coughing procedure where after the upper

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tracheal rings are pinched, the horse not only coughs but groans afterwards. However, this sign is not always elicited.

Another desirable factor in the history is a deterioration in the animal's performance record. He may start the season well but towards the end he goes from win to place to last and finally it is a two week schedule of racing rather than every four or five days. This performance is not, however, specific for "roaring".

Another sign of some value is the occasional "nosebleed", not a hemorrhage but just a few drops of blood at a time. This may be due to increased capillary fragility as a result of anoxia.

Sometimes changes in the humidity may be a factor. A constant question, how does the horse perform on hot, sticky nights; is he worse when racing at night? Does rainy weather affect the breathing? Some of them, by the same token, do well in wet weather while most choke up and never reach the half-mile mark. Occasional, though temporary, relief may be obtained by medicated steam inhalations. After listening to Dr. Churchill, perhaps some of these are cases of chronic pharyngitis and this treatment decreases mucous membrane congestion.

Another diagnostic aid which we use is the recognition of palpable atrophy of the dorsal cricoarytenoid muscle. The finger tips are placed over the top or dosal aspect of the larynx in order to palpate the muscle. A true "roarer" will have a moderate to deep depression or crypt on the affected side when compared with the normal horse which shows no evidence of muscle atrophy. On occasion, following a work-out, one may actually detect fermitus or fine vibrations similar to the sensation imparted to the finger tips when placed over one's own larynx while speaking.

The palpation procedure is simple. With my back to the horse's chest, the left middle finger palpates the dorsal cricoarytenoid muscle. The right ear is placed against the trachea and the right hand is used to turn the horse's head to the left. The left middle finger can sometimes detect increased vibration while the right ear may pick up increased inspiratory stertor. This bilateral procedure is of considerable value though it did take some time to master the technic.

As far as the laryngoscope is concerned, we think it is a wonderful instrument for confirming the case of unilateral paralysis. It is of questionable value in bilateral paralysis for vocal cords may appear symmetrical and often, with the tube in place, the horse fails to breathe deeply.

We do not agree with the common statement in the literature that there is a 90 per cent left side predominance. Perhaps we are dealing with aerodynamics — where the paralyzed left vocal cord creates an air current which bounces off the left arytenoid cartilage and left vocal cord to cause an increased activity of the right ventricule and vocal cord.

Operative Technic

For tranquilization, Promazine in a dose seldom over 250 mg and rarely as low as 50 mg is given. We do not use a twitch nor do we have the head continuously held up in the air. One man holds the shank and slightly elevates the head when required. Of a total of thirty minutes spent on a case, at least twenty are spent on anesthesia. The key to efficient surgery is sufficient local anesthesia to block all pain receptors and maintain a quite animal.

After the usual pre-operative preparation of the site, local anesthesia of the skin and tissues ventral to the larynx is carried out. We often will penetrate the circo-thyroidean ligament and deposit some procaine solution on the mucosa. Perhaps this is not advisable but it gives one an idea as to the depth of the larynx from the skin. We like to see the horse swallow once, not that this is anything abnormal, but it shows what a normal swallowing reflex is like in an unanesthetized animal. Later, we want the swallowing reflex completely abated by our topical anesthetic.

A mid-line incision is made through the skin and carried down to the circo-thyroid ligament. The ligament is incised to the fullest extent, taking care not to damage either the circoid or thyroid cartilages, for it is well known that trauma of these cartilages may cause chondrosis and subsequent biphasic dyspnea.

Rather than a DeVilbiss atomizer for anesthesia of the interior of the larynx and saccules, we use a small spray-type needle which fits on a luer-tip syringe. Spraying with 0.8 per cent Butyn Sulphate is repeated until the area is completely anesthetized as judged by lack of swallow reflex and pressure testing.

Through the laryngotomy incision the unanesthetized epiglottis is touched with the finger tip. In a normal animal the arytenoids will move sharply together and "squeeze" the finger. An absence of this squeezing motion or a difference in degree between the two sides indicates trouble. With regard to Dr. Churchill's remarks on the tension of the vocal cord, if we can slide the finger up towards the second knuckle into a large saccule, there is little doubt about the diagnosis.

With regard to the burr, it should be remembered that the commonly employed Schnell metal burr is a vicious instrument and can create severe damage. It should also be remembered that one can tear out only the base of the saccule. If the burr penetrates the mucosa without inverting it, you have to modify the technic and use Dr. Churchill's method. The burr is to be used lightly. It is advanced to the base of the saccule, turned clockwise or counter-clockwise, and a very slight and continuous tension is put on the handle. For practice procedure, start out by placing a piece of wet Kleenex tissue loosely between your finger and thumb and evert it with the burr. When you have mastered

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the Kleenex, you should be able to strip a saccule completely and safely.

The arytenoid cartilage should not be touched by the burr when the burr is placed in the saccule. This area should be guarded by the index finger. The next step is to gather up the saccule, in the burr as we twist so that it may be everted. Having everted the saccule, the tip of the index finger rests against the now mucosa-covered burr. A curved, 8-inch Carmalt forceps is then introduced into the larynx. With the blades partly open over the shank of the burr, the forceps are advanced over the tip of the index finger and the head of the burr, and the jaws closed over the everted saccule. Even at this stage eversion is incomplete so a second Carmalt forceps is placed behind the first engaged forceps. This may be repeated till it is evident that the entire saccule has been everted.

A pair of curved Metzenbaum scissors is used to follow the convex curvature of the last-placed Carmalt forceps and the saccule severed. We feel that a bilateral operation is usually indicated.

The larynx is very susceptible to trauma and complications are likely unless care is taken during surgery. Generally speaking, we do not use a tracheotomy tube but if surgery is being carried out on a horse with alveolar emphysema, we do insert a tube.

For post-operative treatment we like the horse to stay in a box stall for a minimum of three weeks. By this time the wound will have healed if external applications are kept at a minimum. After discharge from the hospital, a further rest of four to six weeks in the box stall is recommended. Return to work should be gradual and unhurried.

What of our results? From 1952 through 1959, surgery was carried out on 201 cases. Of these, 69 per cent recovered, 4 per cent were improved and 11 per cent showed no improvement. The remaining 16 per cent might as well be placed in the "unimproved, undetermined or unknown" category. These may be cases with dissatisfied owners who turned around and passed the horses to new buyers.

These figures imply then, that three out of ten are going to be unsuccessful in the overall pattern of an unselected group but under some conditions the percentage is lower. We are not very proud of our results. The worst type of animal to work on, in my opinion, is the Hackney or American Saddlebred with a typical "cresty" carriage and resulting laryngeal compression. Our success rate in such cases is much lower. The heavy hunter also may give non-optimal final results in that he is over-conditioned and may make some noise early in the season.

In the unsuccessful cases, where the value of the animal warrants further investigation and surgery, variations on the above described technique have been tried with some success. Upon open-

ing the larynx, if any vestige of a ventricule remains, it is removed with the burr and Carmalt forceps. If a previous unilateral ventriculectomy was done, the often-involved opposite ventricule is removed alone or in combination with the vocal cord. Both vocal cords may be removed in cases where there is no ventricule and the arytenoid cartilage lays wells to the lateral face of the larynx, as well as in cases where only one ventricule has been removed. When the ventricule and vocal cord are removed simultaneously, the arytenoid cartilage seems to hang limp into the lumen immediately following surgery, and yet on endoscopic examination one to two months later, it appears to be back in a normal position.

DR. FOWLER: My discussion topic is a condition commonly called "heaves". Technically it is known as chronic alveolar emphysema. The major portion of the presentation will deal with the clinical aspects of the disease, but I would also like to tell you about research being done to determine the etiology of alveolar emphysema as it occurs in the horse and in man.

HISTORY

Most cases are presented to the veterinarian because the owner has noticed the horse coughing frequently. When the client is questioned about the character of the cough he will mention that it is a dry hacking cough and usually there is no phlegm observed with it. A fit of coughing results when the horse eats hay, especially if it is dusty or moldy. If the horse has been working, the owner will notice a lack of endurance, or after a short period of work the horse will have trouble catching its breath. Sometimes the horse is presented because the horse has recently fallen off in condition. Further questioning will reveal in about half of the cases, the history of an upper-respiratory tract infection within the previous few months.

Certain areas seem to favor the development of "heaves", or at least aggravate an existing case. High humidity tends to aggravate the situation. In California where there are such varied climatic conditions, clients sometimes tell me that the horse is fine at home, but when it is taken to such and such a show it begins to "heave". I recall one case in particular along this line. The horse was a highly trained movie and television performer. He had an advanced case, which would relegate most horses to pasture, but since he earned about $500.00 per week, something had to be done. When he was on location near the ocean, or in altitudes over a thousand feet it was difficult to keep him medicated to relieve the dyspnea, and still have him fit to work. When working at lower elevations and dry climates he needed very little medication.

Also, I have noted that often the horse has recently been purchased. The new owner didn't notice anything with the horse when he bought it, but within 2 or 3 weeks it developed a severe

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cough. These horses make good "trading" horses, especially in early cases, because no symptoms appear until the horse is worked.

"Heaves" is seen in all breeds of horses. It may be more common in the phlegmatic type but it is seen in the occasional race horse. It is a disease of the mature adult

CLINICAL SIGNS

"Heaves" should be considered a symptom and not a disease. A number of signs are found in a typical case of chronic alveolar emphysema; however, none are really pathognomonic. Frequently a practitioner will stand off at a distance and make a diagnosis of "heaves" and with experience, I believe, in many cases this is possible. However, a thorough examination is indicated so that other lung conditions which may be exhibiting similar symptoms are not overlooked.

The principal symptom is dyspnea. Some animals while at rest do not exhibit this to any extent, but following mild exercise a definite expiratory dyspnea may be observed. Associated with the dyspnea is a peculiar movement of the abdomen, called a double abdominal lift, or double expiratory movement. In the normal animal, on inspiration, the rib cage moves outward and the diaphragm contracts, drawing air into the lung. The expiratory phase is accomplished by the elastic recoil of the expanded lung and the relaxation of skeletal muscles involved in inspiration.

In the emphysematous lung the inspiratory phase is normal, but the elastic recoil of normal expiration is absent and expiration is prolonged. Hence, the first part of expiration consists of relaxation of the diaphragm along with relaxation of the rib cage. Since this is not sufficient to expel the residual air, the abdominal muscles then contract to complete the expulsion. The combination of these two actions give the double expiratory movement which, however, is not pathognomonic since it may be evident in other conditions of the lung.

Associated with the abdominal lift, especially in the more advanced cases, is the "heave-line". This is a groove along the coastal arch. In chronic cases it is due to hypertrophy of the abdominal muscles called upon to aid in expiration.

The nostrils are dilated to allow for greater air movement. In nearly all cases there is a non-productive wheezy cough, which is easily induced by exercise or manipulation of the trachea. Examination of the perineal area will reveal a pumping action of the anus. This is caused by the contraction of the abdominal muscles, pushing the intestines back into the pelvic cavity.

In some cases of chronic alveolar emphysema there is intestinal flatulence. The animal may appear bloated all the time. When being ridden or exercised there is an increased expulsion of flatus. The horse usually lacks endurance. If made to work, it recovers slowly from the dyspnea.

Auscultation of the thoracic cavity reveals sibilant or musical rales, especially over the posterior and lower borders of the lungs. These sounds are produced by an accompanying bronchiolitis. In early cases there may be only an increase in the vesicular murmur. If there is a marked bronchitis associated with the condition, one may hear dry rales also. On percussion there is increased resonance. The auscultation area may be enlarged posteriorly due to the expansion of the lung pushing the diaphragm back. Loss of weight associated with digestive upset may be seen in advanced cases even though the animal has a ravenous appetite. The horse may become barrel-chested from the enlargement of the lungs and forced respiration. Along with the forced expiration the animal may arch its spine.

The circulation within the alveolar septa is restricted. The increased peripheral resistance places a greater strain on the heart. To compensate, the right side of heart will hypertrophy. If the condition progresses far enough, a right heart insufficiency or upsets in the conduction system of the heart may become evident and murmurs or skipped beats be heard.

DIFFERENTIAL DIAGNOSIS

Once the initial examination has been completed and a tentative diagnosis made, other conditions which may produce similar signs should be considered. Some of these may be rather quickly eliminated, while with others it is necessary to observe the animal over a period of time to see how it responds to treatment.

Chronic bronchitis is nearly always associated with "heaves". It may also occur independently, but if diagnosed and treated early enough normal lung function will return. The sounds heard on auscultation of a lung with emphysema are those of bronchitis or bronchiolitis. One can get the double respiratory movement and the typical lung sound with a simple bronchitis.

Acute alveolar emphysema may follow diffuse bronchiolitis, an acute spasmodic cough or extensive overexertion. It is also seen in agonal conditions where the animal is struggling for breath. Circumscribed acute emphysema is seen in bronchial stenosis, atalectasis (adjacent tissue), and the opposite lung in unilateral pneumothorax. Acute emphysema exhibits the same symptoms as the chronic type, but the history is different, and if treated satisfactorily the lungs will return to normal, without damage to the blood vessels and elastic tissue.

Another condition is **pulmonary edema** which is seen in equine viral arteritis. It is also encountered in certain types of poisoning, such as 1080 or sodium fluoroacetate, in congestive heart failure, by giving too much fluid intravenously, following surgery when the animal has been recumbent for some period of time, or following strenuous work. Along with the edema there is usually **pul-**

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monary congestion. The two are frequently seen together. These conditions are usually acute and on auscultation and percussion there are differences in lung sounds. There is general venous congestion and there may be a frothy discharge from the nostrils.

Pneumothorax may give an expiratory lift. It is caused by a rupture of the chest wall or an emphysematous bulla. Again auscultation and percussion will differentiate. **Pneumonia** has also to be eliminated. **Laryngeal edema** will cause an inspiratory and expiratory dyspnea. It may be caused by infection or when a carbon disulfide capsule breaks in the pharynx. Palpation with pressure over the larynx will determine if edema is present. **Laryngeal hemiplegia** is characterized by an inspiratory dyspnea, but occasionally the sounds are confused with those coming from the lungs. **Pleural effusions,** following pneumonia must also be considered.

Tumors or granulomatous lesions are among the rare miscellaneous conditions that lead to confusion. Many of these are diagnosed only on post-mortem examination though x-rays may assist in diagnosis during life.

PATHOLOGY

Since "heaves" is a chronic condition and many animals will live for years with it, few opportunities are presented for necropsy. There is however, a very typical post-mortem picture. When the rib cage is opened, it will be noted that the lungs do not collapse, due to loss of elasticity. The lung is enlarged, light and spongy. The borders tend to be rounded. There may be rib impression on the lungs. Also, "air-bubbles" are visible on the surface of the lung. The right side of the heart is usually hypertrophied.

Microscopically there is distention of the alveoli. Some of these may rupture, and coalesce to form a sac or bullae. There is also marked atrophy of the interalveolar septa. The elastic tissue is diminished as are the blood vessels. There is evidence of bronchiolitis in nearly all cases.

ETIOLOGY

The causes of chronic alveolar emphysema and the pathogenesis of the lesion are unknown. Numerous theories have been advanced to explain the symptoms and pathology but as yet, no theory answers all the questions. Heretofore, emphysema has not been produced experimentally so that it could be studied critically.

Some of the theories advanced are:
It is hoped that research now being done will shed considerable light on the causes of chronical alveolar emphysema.

1) Allergy. The allergin causes swelling of the membranes and excessive mucus production. This interferes with the passage of air and leads to emphysema. Bronchial spasm may also be produced.

2) Dusty or mouldy hay, especially when the animal must consume large quantities to maintain its nutritional status. The dust or moulds act as specific irritants, or allergins. Certainly these aggravate the condition but whether or not the action is specific remains to be seen.

3) Chronic coughing, causing such violent expiration that lung tissues are destroyed. A chronic bronchiolitis is associated with nearly all cases of chronic alveolar emphysema. The cough results from irritation of the air passages, and is not the primary etiological agent.

4) Reflected nervous irritation from distended stomach and intestines. This is the theory used to explain the occurrence of "heaves" in animals fed coarse, rough hay. It is felt this causes an irritation of the gastric nerves, which is reflected to the pulmonary branches of the vagus, resulting in a paralyzed condition of the muscular wall of the small bronchioles.

5) Heredity. There is no good evidence for this in the horse, although there are definite familial patterns in the human.

6) Forced exercise. Making an animal extended itself past its endurance for short periods of time, or continued exertion over long periods of time. This is a popular theory. In the days of the draft horse, it was felt that the horse was worked too long and too hard. In those cases where chronic alveolar emphysema follows a period of forced exertion, probably a primary condition already exists. and the exertion merely caused an exacerbation.

The foregoing theories may have some bearing on the cause, but they leave too many questions unanswered. There are however, three other theories being seriously considered today. They are as follows:

1) Obstruction of the air passages. One can see that this may be the primary cause in the allergic response, also in the chronic bronchiolitis type where there is an inflammatory lesion of the bronchioli with excessive mucous plugs. The explanation of this theory is that with partial plugging of the bronchioli, air is inspired because this is the strongest phase of breathing. The air is not expired as fully or as quickly as it is taken in, so a small amount of air accumulates with each breath. Soon the alveolus becomes distended and causes atrophy of the interalveolar septa. Some alveoli rupture and coalesce with adjacent ones. The pressure put on the blood vessels in the septa causes ischemia of the tissue which in turn causes more degeneration.

2) Auto-immune reaction. This is the same type of reaction, (posterior paralysis) that may occur from vaccination against rabies. In this case it is felt that some primary condition of the lung causes tissue destruction. This then acts as an allergen and the body reacts against it.

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3) Interference with the nutritional blood supply to the lung tissue. This causes degeneration of the alveoli and elastic fibers in the septa. The bronchial artery supplies blood to the interalveolar septa in the horse and man. This is not true in all animals, but it is the case in the two species which have naturally occurring emphysema.

A brief review of the circulation of the lung shows that the venous blood in the pulmonary artery is sent to the lung capillaries and is there oxygenated. It is then picked up by the pulmonary veins and is returned to the left heart for distribution to the body. The other system which supplies nourishment to the bronchi, walls of the blood vessels (vasa vasorum) and in the horse and human, the alveoli, begins with the bronchial artery which is a direct branch of the aorta. It follows the bronchi out into the lung where it supplies capillaries to the interalveolar septa. This blood is returned via the bronchial vein, to the vena cava. Even though these are separate systems, there is an anastomosis between the two.

This theory is explained by stating that when the bronchial artery is damaged, the nutrition of the lung is interfered with hence degeneration. This degeneration could cause the commencement of bronchiolitis.

TREATMENT

When it comes to treating these cases, many of us wish they were someone else's problem, because experience tells us that the outcome is usually unfavorable. There are many treatments advocated, and in certain cases we get definite results. However, it is a matter of handling each case individually. If we can retire a horse to the brood mare band, or to pasture, it may live comfortably for years. If, however, the owner wants to continue giving the horse hard usage, treatment frequently is of no avail.

My recommendations for treatment are as follows. Try to avoid hard work. Avoid dusty and/or mouldy hay. Moistening the hay will help. Pasture is very desirable. Feed no ground grains. Avoid bulky feeds. The bronchitis may be treated with nebulizing antibiotics, and perhaps proteolytic enzymes such as Varizyme.

Remission of some of the dyspnea can be accomplished with the use of bronchial dilators. Many agents have been used, such as atropine stramonium, hyoscyamus, lobelia. I have tried a number of these, both in powders and liquid preparation. One of the easiest to use and one that gives good results is fluid extract of belladonna. Eight ounces of the extract is mixed with one quart of distilled water. One to two tablespoons of this mixture is given night and morning on a little grain. One may however, get some undesirable side effects, such as dryness of the mucous membranes and dilatation of the pupil which may cause interference with vision.

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RESEARCH PROJECT ON EMPHYSEMA

Emphysema is one of the most crippling diseases of the human chest. It is becoming more prevalent in young and middle aged men. Since a similarity was found between the horse and human lung, and that both could develop spontaneous emphysema, it was felt that the horse could be used as an experimental animal to reveal the etiology, and perhaps methods of prevention and/or treatment.

Previous to this time, many investigators had tried to produce emphysema in laboratory animals, but had failed, apparently because of the differences in the anatomy of the lungs. A rather detailed study of the comparative anatomy of lungs was made by Dr. Richard McClaughlin, at that time a resident internist in the Navy at Oakland Naval Hospital and Dr. Walt Tyler of the Veterinary Anatomy Department at Davis. They classified lungs into three groups, [(a) cattle, sheep, swine; (b) dogs, cats, and R. monkey; (c) horse and human] based on the type of distal airway and the distribution of the bronchial artery.

In July, 1959, a project was set up to test the hypothesis of bronchial artery disease. It is administered jointly by the Clinical Investigation Center of the Office of Naval Research and the University of California Veterinary School at Davis.

RESEARCH TECHNIQUE

The effects of the injection of a sclerosing agent into the bronchial artery are being studied. The animal is restrained in lateral recumbency and the thoracotomy approach can be made from either side. The sixth or seventh rib is removed and the bronchial artery isolated as it leaves the aorta on the right side or as it enters the hilus of the left lung. The artery is either injected at that time, or a cannula is placed in it and after the horse has recovered from anethesia the material is injected.

Artificial respiration is a problem in open chest surgery. We developed our own system for use on the horse. It is comprised of a Bird Respirator connected to a tank of oxygen and a tank of nitrous oxide. The valves on the oxygen and nitrous oxide tanks had to be bored specially to allow for the increased flow necessary. Intercallated in the system between the respirator and the horse are three etherizers and a pair of exhaust valves. A cuffed endotracheal tube is inserted through a speculum in the mouth. The system is a semi-closed one. One of its major disadvantages is that it does not make efficient use of the gases since no arrangement is made for rebreathing, thus it is rather expensive to operate. However, it gives excellent positive pressure breathing, and it is not necessary to have some one sit at the machine constantly and squeeze a bag. We have used this system on a horse weighing 1,500 lbs.

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The sclerosing agent used is chlorpromazine or Thorazine. When given intra-arterially in dilutions as low as 1 to 25, it causes an endarteritis.

The technical problems associated with open chest surgery in the horse are many. The correct titration of a dose of Thorazine that will do the desired damage but not destroy the lung to the point where the animal dies, has been difficult. We have lost a number of horses within 5 days of surgery from this cause.

Fourteen horses have been operated upon with results as follows: One animal died on the table from heart failure. On another occasion the heart stopped, but started again following cardiac massage. Four horses never regained their feet after surgery. This was probably the result of shock plus the fact that we gave large doses of Thorazine in the earlier cases and the damage to the lung was too marked. Three animals recovered from anesthesia but died from acute pulmonary congestion and edema within 5 days. Post-mortem examination revealed what appeared to be acute damage to the bronchial artery caused by Thorazine. Three horses lived for 3 weeks. One of these had an esophageal slough as a result of Thorazine getting into the esophageal branch which comes off the bronchial artery; one had a surgical infection and the other is presently living, 3 weeks after surgery. Two lived for three months. One had a chronic infection, the other developed emphysema.

One animal is alive today, 16 months following surgery. This was one of the first animals operated upon, and it is possible that the Thorazine was placed in a bronchial vein instead of the artery, as this horse shows no evidence of heaves.

Our future plans are to perfect the experimental production of emphysema using the bronchial artery, then to test the autoimmune hpyothesis and the obstruction of air passages. We also plan to do extensive pulmonary function studies on these horses which should give information on respiration in general in the horse.

Our findings to date are as follows: There are apparently two different conditions in the horse. One is the typical "heaves" associated with chronic bronchitis. The other is a primary emphysema not associated with bronchitis which does not reveal any abnormal sound on auscultation. We have observed this at surgery. There is a great deal of reserve lung tissue and unless half or more of the lung is involved there would be no dyspnea, hence the only way this could be detected is by less endurance or poorer performance. This fact makes one wonder how many cases of emphysema are unrecognized in horses.

We also believe that we have produced emphysema experimentally by sclerosing the bronchial artery.

CHAIRMAN TEIGLAND: Are there any questions?

DR. WISEMAN: Some lady back in the east, in Massachusetts I believe, has supposedly become quite an authority on this sort of thing and developed a feed. I wonder if you looked into that.

DR. FOWLER: I know of the lady but don't know her personally, but I have read the article. I was a little bit surprised to read it in a professional publication that came out of the New England States.

I am always sketical when somebody says, "I have taken 20 horses and I have cured all 20 of these horses. I have had no relapses. I have hundreds of people that swear by my feed".

Benefit is claimed for many feeds but I question this. I think routine husbandry methods are worthwhile and may definitely improve some of these cases. I cannot discount the possibility of an allergic reaction. This is what she is basing her cause on, but I believe we need some more basic research.

DR. O'DEA: We have a problem in the east which I think, because of our climatic conditions, is more acute than your situation is in the west. Those of us who practice in the east, particularly on farms where they make their own hay, know that in the years when we have the highest incidence of the classical heaves, we have had a wet June.

We had to be interested in it (hay-free diet) because of the incidence of heaves and because of the value of the horses involved. Being interested, as a man in practice who has to make money, we approached the problem from the standpoint of allergy. All the symptoms of an allergy were present — an asthmatic syndrome as seen in the human. It was a case of whether the allergy was a matter of pollens or a matter of molds, so, we had pollens produced and, also, in the laboratory, the full range of molds that were evident in moldy hay.

Into sealed box stalls were placed different groups of clinically normal animals. In some stalls pollens were circulated and in the other stalls we circulated the molds. In the stalls in which we circuated the molds, we definitely did find the production, over a period of days and weeks, of actual bronchitis with high-pitched crepitant rales, dry cough, etc. As the process continued, the heaves got worse and a heave line developed.

Dr. Bill Wright down in New York City has been doing a lot of work with the hay-free diet. The point of the hay-free diet is the matter of getting away from the mold. When a barn is dusty or when the horses in the next stall are having hay, and the circulation of molds is too high, we see no change or improvement in the allergy and, consequently, in the heave syndrome, even when on a hay-free diet. If, however, the individual is isolated, and then put on the hay-free diet, the improvement is absolutely amazing.

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I think once coalescense of the alveoli has occurred, the outlook is bad. If treatment commences in the early stages, before there is coalescence of the alveoli, and where there is a true allergic situation, then improvement can be expected and you can convince that owner of the necessity of having proper roughage, and at the same time get away from the moldy hay. I think a tremendous amount of good is actually being accomplished by the hay-free diet. If you have proved nothing else, you have proved to the owner that he has to have good feed.

DR. FOWLER: Certainly, it could be a combination of factors that is causing this, and the allergy could cause the air obstruction. I believe your point is well taken.

DR. O'DEA: What is your finding in the use of aminophylline?

DR. FOWLER: I have not used aminophylline. I have used belladonna and stramonium. They seemed to give me what I wanted.

DR. CHASSELS: Have you used Fibromin? If so, what results?

Dr. FOWLER: I don't believe I am acquainted with the product. Would you like to comment?

DR. CHASSELS: I have used it quite a bit. Dr. Catlin of the New York Racing Association suggested it to me. I have had fairly good results with it in some cases. I have no idea how it worked or why it would work, but I think it might break up some of the congestion in the lungs. The dose is 10 cc. every two or three days, for a total of 30 cc. Has anybody else used it?

DR. FOWLER: I might just mention, if it breaks up the exudate within the bronchioli, I can see where its use is valid. As such, in heaves there is no scar tissue within the lungs. We have a lack of scar tissue rather than abundance of it.

DR. GUARD: Dr. Delahanty said he was not satisfied with his results in the roaring operation, and he mentioned that he always does what I consider in one respect to be a violation of the best principles of surgery. When one operates on the normal side, I do not think the arytenoid cartilage and vocal cord will be pulled sufficiently to the side. There will be some "filling in" and he will be left somewhat thick. Perhaps his results would be better if he operated only on the affected side.

DR. DELAHANTY: Dr. Guard, in those cases where it is a clear-cut left side paralysis, we don't touch the right saccule.

DR. GUARD: I thought you said you routinely operated on both sides?

DR. DELAHANTY: I haven't found one or two in the last 200 where the right side was normal.

DR. GUARD: I don't know how you determine it!

RECENT ADVANCES IN THE DIAGNOSIS OF EQUINE HEPATIC INSUFFICIENCY

CHARLES E. CORNELIUS

An interesting and long-standing observation among equine practitioners has been that icterus is a fairly common clinical symptom in the horse, but diagnosed hepatic disease is rare. This finding most likely results from common duct closure and obstruction secondary to intestinal inflammation; however, there has been no experimental vertification of this theory.

Bile pigments result from the rupture of senescent erythrocytes[1] and the subsequent reduction of heme in cells of the Reticulo-endothelial (RE) system. Free or unconjugated bilirubin is liberated from the RE cells and subsequently conjugated by the liver to a soluble diglucuronide[2] or sulfate prior to excretion into the bile (Fig. 1). This more soluble conjugated bilirubin will not precipitate during the excretory process. The historical van den Bergh Test when performed on all species except the horse, allows the clinician to distinguish between the free bilirubin (indirect-reacting) resulting from rapid erythrocyte hemolysis and the soluble bilirubin diglucuronide (direct-reacting), which is conjugated in the liver and regurgitated back into the blood due to either intrahepatic or external bile duct obstructions. Unfortunately, most all of the bile pigments recovered from the serum of horses in obstructive icterus (hepatic or extrahepatic) have been up until now, chromatographically identified as free bilirubin (indirect-reacting) and not the conjugated bilirubin as expected and observed in association with similar pathology in other species. In only one horse have we found conjugated bilirubin in an appreciable concentration. It is not known at present why the regurgitated bilirubin glucuronide in obstructive icterus loses its glucuronic acid fraction upon entry into the blood of the horse. Preliminary evidence indicates that the van den Bergh test is of little help in distinguishing the cause of icterus in the horse.

Total serum bilirubin levels have been observed to range between 0.2-6.2 mg/100 ml with a mean concentration of 2.7 mg/100 ml[2]. Considerable variation in the serum bilirubin level occurs due to changes in diet or from fasting[3]. Bilirubin levels from acute liver damage may reach 15 mg/100 ml serum, while hemolytic crisis will cause elevations as high as 30-50 mg/100 ml.

*Hynson, Westcott, and Dunning, Inc., Baltimore, Md.

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Non-hepatic Causes of Icterus

1. Hemolytic anemias, e.g. Infectious viral anemia.
2. Colic or intestinal stasis.
3. Starvation.
4. Encephalomyelitis with intestinal complications.

Hepatic Causes of Icterus

1. Plant poisons, e.g. Amsinckia or Senechio species.
   These weeds produce a diffuse fibrosis of the liver with terminal nervous system (CNS) symptoms. There is no evidence at present that the CNS involvement is similar to the Hepatolenticular Syndrome in humans (Wilson's Disease with Ceruloplasmin deficiency). The symptomatology of equine hepatic insufficiency with CNS involvement is probably similar to that observed in chronic alcholic hepatic fibrosis with terminal coma observed in man.
2. Liver abscesses with progressive peripheral hepatic necrosis and fibrosis. Terminal CNS symptoms have been observed in three cases recently associated with diffuse hepatic involvement.
3. Hemosiderosis — observed following severe chronic hemolytic disease.
4. Acute anemia — produces hepatic necrosis from rapid bacterial growth in the liver.
5. Malignancies — from metastasis.

Differential Diagnosis

Occasionally icteric horses are presented for diagnosis with central nervous system symptoms such as incoordination, staggering or paresis. A few simple and readily available liver function tests will allow the clinician to distinguish between (1) a primary involvement of the brain (such as encephalomyelitis or tumor) with secondary icterus from intestinal complications and (2) a primary hepatic insufficiency with secondary CNS symptoms from abnormal levels of circulating metabolites. Rectal palpation may occasionally be of aid in determining the size and consistency of the liver.

Liver Function Tests

1. **Total serum bilirubin level** — if the serum bilirubin level is over 6 mg/100 ml. (in the absence of hemolytic disease,

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determined by using the Wintrobe Hematocrit for packed cell volume), intraphepatic obstruction of biliary canaliculi associated with hepatic cellular disease is most likely present. Extrahepatic bile duct involvement is quite rare.

2. **Bromsulphalein (BSP)*** — this dye is available in 1 gram ampules and 1 gram is injected intravenously. Following an approximate 5 minute period for dye mixing, 2 blood samples are withdrawn 4 **minutes apart** into heparinized syringes. The two samples should be taken between 5 and 12 minutes, e.g. 5 and 9, or 6 and 10 minutes post injection. The plasma BSP concentration in the second sample should normally always be at least **one-half** the concentration found in the first sample. Semi-log paper may also be used as shown in Fig. 2[5]. $T^{1/2}$ values for dye clearance in normal horses ranged between 2:0-3.7 minutes[4]. All commercial laboratories determine serum BSP concentrations routinely. It is advisable, however, that 2 ml of plasma be used by the laboratory in the case of the horse rather than the routine 0.5 ml as used in man. Both samples are diluted to 6 ml with a dilute NaOH solution before reading. Delayed excretion of the BSP from the blood may be due either to hepatic fibrosis or necrosis. Severe dehydration or cardiac decompensation may produce a lowered hepatic blood flow and also delay BSP removal.

3. SGO-T (Serum Glutamic-oxaloacetic Transaminase). This enzyme is found in high concentration in the liver, and skeletal and cardiac muscle. Normal serum levels in the horse usually range between 120-200 units/ml regardless of the method performed by the commercial laboratory[6]. In the absence of myopathies (dystrophies, tying-up, or Azoturia), elevations in the serum enzyme activity will indicate the presence of hepatic necrosis and the release of the enzyme from hepatic cells to the blood (Fig. 3). Horses with severe clinical hepatic necrosis may exhibit serum levels of SGO-T activity above 1000 units/ml. Horses with "scarred" livers, free from necrosis, will have normal SGO-T levels with delayed BSP clearance. The use of the two tests simultaneously will determine the nature of the hepatopathy.

4. IC-D (Isocitric Dehydrogenase). This enzyme is also present in cardiac and skeletal muscle and liver of horses. In experimental hepatic necrosis as well as in clinical cases, this enzyme is also liberated similarly into the serum as in the case of the SGO-T. The normal level of IC-D activity in mature horse plasma is 600 ± 200 units/ml with a range of 291-1075 units/ml[7].

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DR. COSGROVE: What dye is used? The name was not mentioned, I believe.

DR. CORNELLUS: Bromsulphalein, (BSP), and the drug company is Hynson, Westcott & Dunning. There has been no adverse reactions so far, although perivascular infiltration will cause a sloughing reaction.

DR. SMITH: Is there any value in a cephalin flocculation test in the horse?

DR. CORNELIUS: Using something like a serum dilution of 1:1000, one individual worked up the cephalin flocculation test for the horse. It is much better to send it to the laboratory for a simple albumin determination. Don't forget the horse may have an A/G ratio as low as 0.4. The horse has the smallest amount of albumin of any common domestic anical. This is why edema is so commonly found in horses; because of this normally low albumin-to-globulin ratio.

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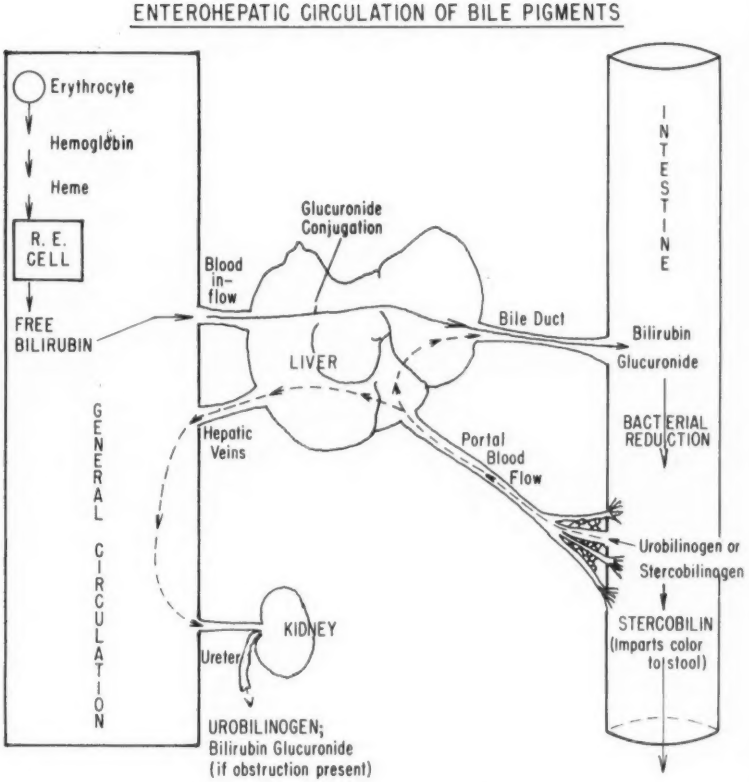

Fig. 1. Enterohepatic Circulation of Bile Pigments

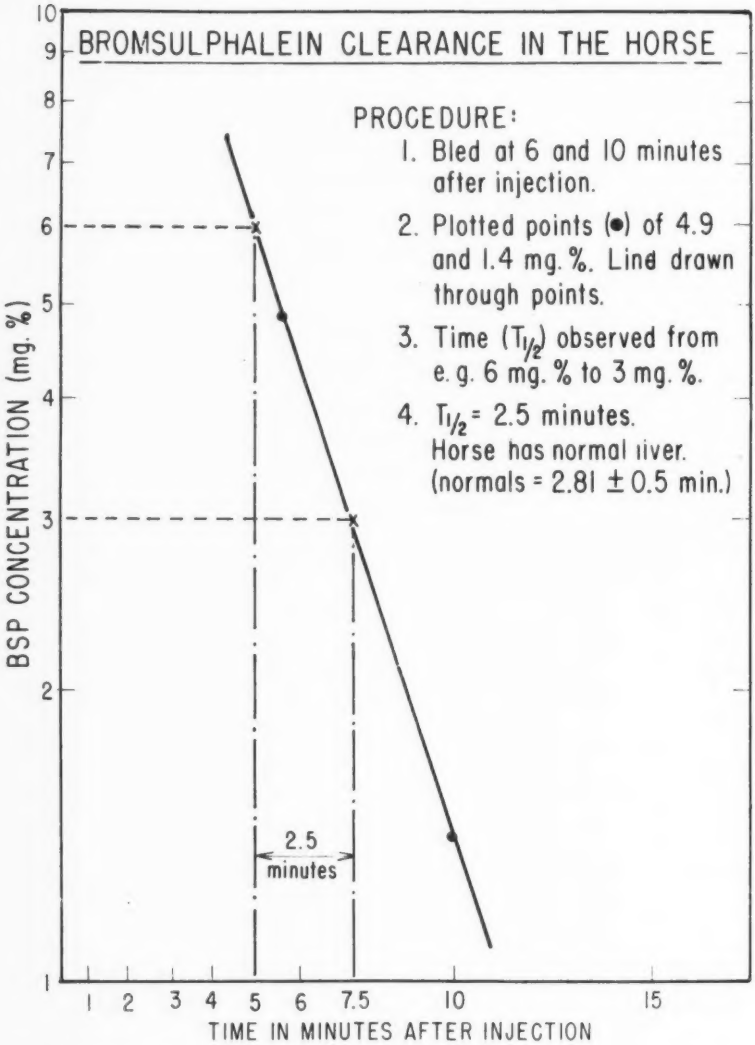
BROMSULPHALEIN CLEARANCE IN THE HORSE
PROCEDURE:
1. Bled at 6 and 10 minutes after injection.
2. Plotted points (•) of 4.9 and 1.4 mg. %. Line drawn through points.
3. Time ($T_{1/2}$) observed from e. g. 6 mg. % to 3 mg. %.
4. $T_{1/2}$ = 2.5 minutes. Horse has normal liver. (normals = 2.81 ± 0.5 min.)
BSP CONCENTRATION (mg. %)
10
9
8
7
6
5
4
3
2
1
2.5 minutes
1 2 3 4 5 6 7.5 10 15
TIME IN MINUTES AFTER INJECTION

Fig. 2. The use of semi-log graph paper in calculating the $T^{1}/_{2}$ for Bromsulphalein (BSP) disappearance from the serum.

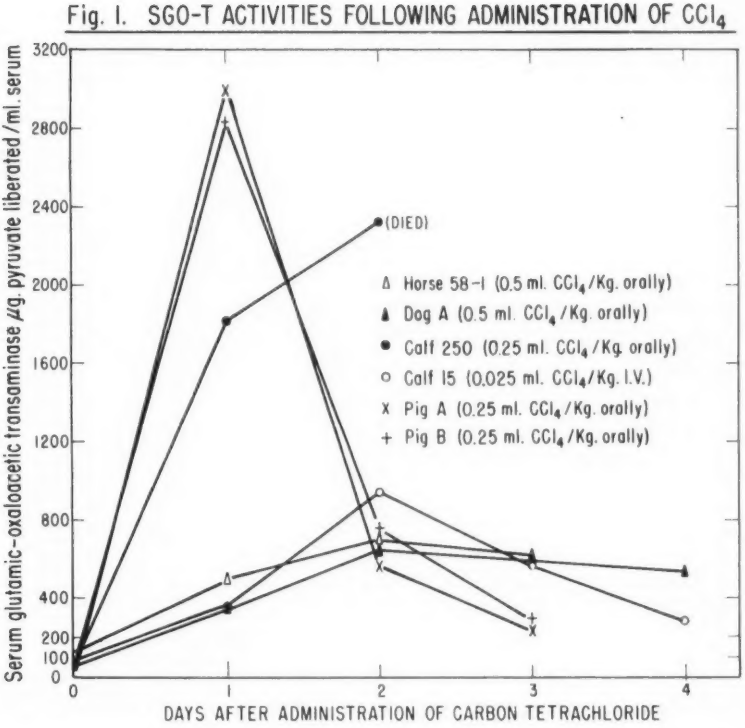

Fig. 3. SGO-T activities following Administration of CCl_4[6]. Note significant elevation in the horse.

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PANEL ON ORTHOPEDIC SURGERY

Moderator: M. B. Teigland

Panel: Jacques Jenny
J. D. Wheat
D. L. Proctor
W. O. Reed

MODERATOR TEIGLAND: The orthopedic surgery panel which has been a yearly feature of our annual meeting will be opened by Dr. Jacques Jenny whose topic is "Pathogenesis of Bone and Joint Disorders".

DR. JENNY: The annual meetings of associations such as the AAEP play an important part in our continuing postgraduate education. Panel discussions, illuminating the same general topic from different viewpoints, have become particularly popular. During the past two years, some very radical new approaches to old problems have been offered. Syringe and scalpel are now invading areas which ten years ago were absolutely taboo. While technical aspects of new procedures have been stressed, a review of newer concepts concerning the structure and function of the parts involved has so far been neglected. I have been asked to summarize some basic information as a background against which our modern therapeutic measures could be critically evaluated.

Most of the bone and joint leisons confronting the track veterinarian are the direct or indirect result of a single trauma or the summation of stresses to which the racehorse is subjected. If a 1000 pound horse carries a 110 pound jockey in 34 seconds over ⅜ of a mile, it produces a kinetic energy ($m/2\ v^2$) of 1,867,020 foot pounds. It should be pointed out that in our calculation torsion forcess are not considered, that the stresses are repeated, and that the subject is frequently an immature animal. If we tell a structural engineer that this tremendous thrust is received by a galloping horse on one single front leg, a bony column of scarcely 3 inches in cross section, he will tell us that there is no man-made structure of similar proportions which could withstand such forces. What enables the horse to withstand these stresses? The secret is an exquisite shock-absorbing mechanism and the faculty of all living tissue to continuously regenerate. In the angulation of the fetlock, in the muscles, tendons and ligaments controlling its position, in the minute details of the hoof, in the ingenious arrangement of joint surfaces and their lubrication system, we find a wealth of details which could never be included in a man-made device conceived for this purpose. Of course such an intricate system offers a multitude of possibilities for breakdown. Use-destruction, wear and tear, is inevitable especially if the system is taxed by peak performance as is demanded of a racehorse.

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Again, living tissue is capable of withstanding wear and tear to a much high degree than is the case with inanimate material. If a metal bar is subjected to repeated bending stresses, it will ultimately show a fatigue break. Bone on the other hand can resist such forces almost identifinitely since osteoblasts and osteoclasts continuously tear down bone substance and rebuild it to conform with stress patterns. If an intramedullary nail, employed to stabilize a transverse fracture of the femur, is left in the marrow cavity after the fracture has healed, the nail can suffer a fatigue break as the limb is subjected to the normal, repeated stresses of activity, while the bone around it remains intact. Fatigue fractures in bone occur only if the repeated stresses exceed the rate of tissue repair. This replacement of "used-up" substance is characteristic of all living tissue within the limits of quantitative and qualitative variations. As supervising engineers or repairmen it behooves us to know the regenerative capabilities of different tissues if we want to prevent or correct bone and joint disorders. Generally speaking, we can claim an intimate familiarity with muscle, tendon, ligamentous and bone tissue, if our memory reaches back to the anatomy lectures of our college days. Many new concepts concerning the details of structure and function of joints and their defense or repair mechanisms have appeared in the literature of the last ten years. These are more or less in step with the advances made in surgery of these areas. Using my initial sentence as a form of apology I should like to take you back to the classroom.

The joints which concern us therapeutically are classified as synovial or diarthrodial joints. These permit more or less free movement of the adjacent parts and are characterized by the following structural elements:

(1) The two opposing joint surfaces are covered by hyaline cartilage.
(2) They are joined together by a fibrous capsule and ligaments.
(3) The inner lining of the capsule consists of a vascular connective tissue which produces.
(4) The synovia or joint fluid, forming a lubricating wedge between the cartilaginous surfaces.

Let us again make an analogy with a man-made counterpart. To minimize friction, a mechanical joint requires periodic greasing or, if it is a more modern greaseless joint, replacement of the contact surfaces after a certain period of use. Neither is necessary in a diarthrodial or synovial joint. Normal wear and tear can be offset by tissue regeneration; even the lubricating joint fluid is continuously regenerated. Let me review the mechanical features and then the histological aspects which ensure continued "trouble-free" function of a horse's joints. To minimize friction between the mobile elements, the joint surfaces are incongruous. (SKETCHES ON BLACKBOARD). Male and female components

do not match so that cartilage to cartilage contact is at a minimum, even at rest. During motion this incongruity of the joint surfaces acts as a pump and builds up pressure within the lubricating fluid. This pressure is great enough to hold the opposing surfaces apart, so that there is no cartilage to cartilage contact while the joint is in motion. The effects of friction on cartilage are therefore maximal at the beginning and at the end of movement. It is obvious that the quality of the joint fluid is of prime importance to the well-being of the joint, especially during the initial and end phase of motion; the more viscous a lubricating fluid, the greater its ability to resist shearing forces and to compensate for a relatively slow rate of movement. The viscosity of synovial fluid is almost entirely due to the non-sulfated mucopoly-saccharide, hyaluronic acid. Otherwise it can be considered as a dialysate of blood plasma. It also contains, normally, a few cells derived from the lining tissue. These cells are mononuclear and rounded since they are free swimming in the intra-articular space. Inflammation of the synovial membrane can alter the cellular content of the joint fluid.

Let us have a closer look at the synovial membrane. It is a connective tissue, the cells of which are indistinguishable from connective tissue cells elsewhere. Its most characteristic morphologic feature is a capillary network adjacent to the joint cavity. A variable number of folds and villi project from the surface of the synovial membrane, which is otherwise relatively smooth. In addition to the cells, fibres, and blood vessels it contains lymphatics and few nerve fibres. Most joints also are equipped with fat pads without any special form or structure. Gardener (1) compared them to what engineers call "pad oilers", a little pad with oil on it that is used to wipe a bearing surface.

Articular cartilage is an avascular, nerveless and relatively acellular tissue. It is resilient and elastic. The deeper layers are nourished from capillary tufts in the sub-chondral plate, the superficial layers from the synovial membrane through penetration of joint fluid. Ekholm (2) has shown that tracer elements enter the cartilage from both vascular beds and that the rate of entry is increased with exercise. The water content of the surface layers is increased with use and decreased at rest in amounts sufficient to vary the thickness of the cartilage as much as ten per cent. Histologically the hyaline cartilage can be sub-divided into three layers (SKETCH ON BLACKBOARD),

(1) a deep, calcified zone, the subchondral plate,
(2) a broad mid-zone of high water content and great shock-absorbing capacity and
(3) a thin tangential fibrous zone on the surface.

The older literature maintained that adult cartilage has lost most of its power of growth and repair. A recent Swedish publication (3) demonstrates a rather active cartilage turnover in the adult.

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Regeneration occurs by cell proliferation in the upper mid-zone in form of clones or "brood capsules". These cell clusters reach a maximum in about three weeks and become separated from one another to re-establish the normal cell-matrix pattern in the following ten weeks. Cartilage which has been subjected to vigorous exercise may be 25 to 45 per cent thicker than "rested" cartilage. In weight-bearing areas it is normally thicker. Excessive loads can thus produce localized regions of excessive cartilage proliferation. If this proliferation exceeds the shedding rate, the cartilage thickens. Cartilage thickness can reach a critical limit if the penetration of nutrients from the surface and the subchondral plate can no longer reach the mid-zone. As this occurs, cartilage will degenerate and with the appearance of a bloodvessel, a center of ossification will be established. Thus a joint surface can be remodeled according to the demands of specific stresses, conformation and/or excessive loads. Johnson (4) has demonstrated the kinetics of such joint remodeling processes and different forms of marginal lipping (BLACKBOARD SKETCHES). The line where these adaptation-to-stress changes become pathologic changes are difficult to draw.

Johnson (4) pointed out an even more intriguing form of adaptation to specific demands in the adjustment of the viscosity of the synovia for different joint activities. The matrix of cartilage contains a sulfated mucopolysaccharide, one of the chondroitin-sulfuric acids. As was pointed out earlier, the cartilage of the broad mid-zone becomes, in the course of the shedding process, the cartilage of the tangential zone. In the upper mid-zone, just beneath the tangential surface zone, the cartilage is eosinophilic, indicating loss of sulfate. Desulfating the mucopolysaccharide makes it a possible precursor of hyaluronic acid. The movement of water in and out of this surface zone with exercise, as Ekholm demonstrated, offers a mechanism for leaching out the polysaccharide. Thus the degree of viscosity could indeed be correlated with the activity of the joint.

The limited time I dare usurp from my fellow panelists does not allow a discussion of the actual pathogenesis of many joint disorders. I should hope that the preceding review would stimulate you to take a second look at some of the joint pathology you see every day. According to Churchill (5) "osselets" are caused by the anterior border of the first phalanx hitting the corresponding area of the third metacarpal bone. I should like to submit that the build-up of pressure within the joint capsule is the inciting cause. As the fetlock approaches a 90 degree angle the incongruity of the opposing joint surfaces forces a fluid wedge into the anterior portion of the joint capsule. Under this repeated, extreme pressure the joint capsule of the still-immature horse distends and then thickens. The progressing fibrosis of the capsule will make it strong enough to withstand the pressure and the points of at-

tachment become the areas of least resistance. The appearance of calcium or rather, new bone deposits at the joint capsule attachment is therefore the third stage in this sequence of events. The practical implication of this theory is very obvious and can be repeated for virtually all joint disorders in racehorses. The built-in safeguards can become harmful if over-taxation of a joint is prolonged. The anterior border of the third carpal bone is subjected to extreme pressure as a horse comes down the home stretch. This pressure results in a cartilage build-up and ultimately in marginal lipping through remodeling of the joint surface. If this remodeling were allowed to progress undisturbed, once it has been set in motion, no harm would come to this joint, however, continuing stress, interfering with this remodeling process, results in either a rough, anterior joint-border or a chip fracture.

The pathogenesis of most bone and joint disorders follows the same pattern. It is initiated by the inability of the regenerative capacities to keep up with use-destruction. It is then aggravated through interference with the repair pattern by continued microtrauma instead of rest. I should add that a horse with poor conformation subjected to normal stresses and one with perfect conformation subjected to excessive stresses will show principally the same wear-and-tear pathology.

If my review has re-kindled your respect for the delicacy and intricacies of joints and cautioned you against indiscriminate invasion of these structures with syringe and scalpel, my effort will have been worthwhile. Since I compiled the data for this paper I cannot help but think how much cartilage will be needed to reconstitute the proper viscosity of the joint fluid each time I drain a joint capsule during the exposure of an articular facture. Would I dare to aspirate the synovia prior to surgery and reinject it after closure of the joint capsule?

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MODERATOR TEIGLAND: Are there any questions to be asked of Dr. Jenny? It might be well to ask them while they are still fresh in mind.

DR. BURCH: Is there any way that one can distinguish, radiologically, the normal, abnormal and fractured meniscus in the stifle joint?

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DR. JENNY: The only way is by estimation of the joint space. An A.P. radiograph of the equine stifle can be quite difficult especially if it has to be made at the track, on account of the possible drop in line voltage. Possibly the insufflation of air might help, but unless the horse is very sick, I doubt if you would want to do that.

DR. PHILLIPS: I would like to ask Dr. Jenny the mode of action of procaine and why sometimes it seems to last three or four weeks and other times only a few days.

DR. JENNY: I assure you, I can only speculate as to the proper answer! One of our colleagues here told me yesteday that he injected a horse's joint for diagnostic purposes exactly as it was shown in the movie we saw at our meeting last year. He injected 10 cc of 2 per cent Xylocaine solution and ended up with an almost stiff joint. The first thought in his mind was infection but cultures of joint fluid were sterile. Hence there are only two explanations open: (1) The joint fluid was diluted to the point where its viscosity became nil. To reconstitute the normal viscosity an excessive amount of mucopolysaccharide was "leached" out of the mid-zone of hyaline cartilage and as the vertical, not only the tangential fibres, of the cartilage become bared, the joint was on its way to progressive osteoarthritic changes. (2) The hyaluronic acid can be destroyed by precipitation with an acid. Whether or not the large amount of Xylocaine might set up such as inflammation that the ph would become acid and thus destroy the hyaluronic acid I would not know, but I'd consider it a possibility.

MODERATOR TEIGLAND: The next speaker is Dr. J. D. Wheat of California who will discuss "Contra-indications in Bone and Joint Surgery".

DR. WHEAT: Since, even without treatment, very few sesamoid and carpal fractures will not make a satisfactory enough recovery for a filly to become a brood mare, or a colt a breeding stallion, it is apparent that almost the only reason for surgical intervention is to enable the horse to return to racing.

The first consideration is the owner's plans regarding the future of the horse, for instance I have operated successfully on the occasional horse only to have the owner say "I had planned to make a brood mare out of the filly anyway, so I think we will have her bred rather than try to race her again". This is more common in the case of a filly than a colt.

Other factors for consideration are the estimated racing life of the horse, sex, value and general condition and conformation of the other joints and the opposite leg. A horse should be capable of another two years of racing. It must also have enough racing ability to warrant the cost of surgery, after-care and an extended rest period, the total cost of which will be $2000. or more before the horse is ready to compete again.

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Although the sex of the horse is not a major reason in reaching a decision of whether to operate or not, nevertheless, it does have to be considered. A big, heavy-bodied, hard-to-handle stud does not merit as favorable a prognosis as a light-weight, more agile horse that is going to put less wear and tear on its legs.

The owner's acceptance of surgical intervention is important. If he is reluctant to have the horse operated upon or is sketical of the outcome, I do not believe that it is a good idea to persuade him otherwise. The main consideration is the actual condition of the affected joint. If there is any indication or suspicion of infection as shown by cloudy synovia, fibrin flakes in the synovial fluid and excessive heat, pain and swelling, surgery should be delayed or avoided entirely. If, due to injury, excessive joint swelling is present, I prefer to wait until reduction of the swelling is effected, thus avoiding the problem of increased vascular oozing and tissues which do not yield readily to retraction and manipulation.

Not every horse with exostosis or fracture of the carpus is a suitable surgical subject. After operating on a considerable number of knees without eliminating those that looked unfavorable from the standpoint of chronicity and excessive degeneration, I have observed that the only cases which offer a high degree of success are those that have a bone chip which is free of the carpal bone from which it originated and are not accompanied by secondary exostosis and other degenerative changes.

Surgical procedures on the fetlock joint are concerned primarily with sesamoid fractures and the removal of free bodies that originate as fragments from the articular margin of the first phalanx. Here, as with carpal fractures, the free bodies along the articular margin should not be associated with secondary exostosis as the result will be continued lameness even though the free chip has been removed. Sesamoid fractures should not be operated upon if the line of separation is less than one eighth of an inch. Furthermore, good results should not be anticipated if a large portion of the sesamoid bone has to be removed. I hesitate to operate on a fracture that involves more than 20 per cent of the total dorsoventral thickness of the sesamoid bone.

Surgery on old fractures does not give good results. The degenerative changes, exostosis and fibrosis which develop are a great hindrance to a successful outcome.

If dorsal luxation of the sesamoid bone has occurred, no value is to be gained by surgical intervention.

Following fracture of the ventral aspect of the sesamoid bone, some horses develop a fibrosis and contraction of the distal sesamoidean ligament with knuckling of the pastern joint as a result. Removal of the fractured portion will not correct the contraction of the ligament.

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MODERATOR TEIGLAND: Are there any questions for Dr. Wheat at this time?

DR. TRUSSELL: Are you of the opinion that heredity plays any part in some of these cases?

DR. WHEAT: Conformation is certainly a major factor in chip fractures. Most of the cases which we see are of weak-kneed or calf-kneed conformation and this predisposes to carpal fractures.

DR. C. H. REID: In the instance of a fractured sesamoid of two years' standing, with the fragments an eighth of an inch apart posteriorly but apparently joined in front, would you consider that that animal could be brought back to the track by an operation? This is not a horizontal fracture; it is wedged-shaped.

DR. WHEAT: In such a case, you would have considerable difficulty in separating the fragments. By this time there would be a quite firm union between the pieces. Surgery would be contraindicated, in my opinion. Unless such cases are operated upon early, I think one does more damage than good by going in and separating the areas of fibrosis and exostosis. The recovery rate of these cases, as far as racing ability is concerned, is very low.

DR. COSGROVE: The type of fracture we see in Ireland and England is confined mainly to the pisiform bone where vertical and horizontal fracture lines are noted. Is there any type of surgical intervention that would give a reasonable racing prognosis in such fractures?

DR. WHEAT: I have not attempted surgery on such cases. We see very few of this type of fracture. The only suggestion I can make is the use of a nail or screw to hold the fragments in place.

DR. REED: The fracture which Dr. Cosgrove is referring to is that of the accessory carpal bone. We have had some results when a considerable amount of time was given to the horse, followed by neurectomy. One horse became a jumper one year after such a procedure. I think you might find this type of fracture occurring quite frequently in top jumpers over there.

DR. JENNY: I would like to ask Dr. Wheat what he would do with one of those small chip fractures that I demonstrated!

DR. WHEAT: I recently examined such a case. The horse had been laid up for about a year and a half. When placed back in training, it was still lame. The owner gave up in disgust and sold the horse. At the new owner's insistence, I opened up the joint as an exploratory procedure. There was a band of calcification in the synovial membrane. This was dissected out and the synovial membrane closed by sutures. At the present time, the horse is progressing quite well. I do not know what the final outcome will be. There is a horse which had at least one and a half year's rest and was no better than when he was laid up. X-rays of this type of pathological change reveal an obvious thin or fine area of

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calcification but on opening the joint the lesion is much more extensive than it appeared on the plate. Also, on a lateral view, it is difficult to determine the lateral-to-medial extent of the lesion. On the film, it may appear, in cross section, to be a small, thin fragment but invasion of the joint may reveal a quite considerable lesion. The same effect is seen in fractures of the third carpal bone. The reason for this, is that it is hard to outline the fragment on an anterior-posterior projection. In some cases, it is advisable to carry out an exploratory examination of the joint, for the findings often provide valuable information, particularly in those horses that have been given sufficient time to make a recovery. On some of these cases we have used x-ray therapy and have been able to get them back to racing.

DR. PROCTOR: In practice, when I say "this is an apical sesamoid fracture" or "this is a chip off the radial carpal" or "this is a slab fracture of the third carpal", the owner wants to know what the chances are of that horse being an economic success. I would like to have a meeting of minds of the three other panel members and have them come up with a "success percentage" following surgery.

MODERATOR TEIGLAND: In contacting these men for this part of the program, I asked for this specific answer to be included. When I talked with Dr. Raker he said that it was very difficult to make such an estimate because as far as the universities are concerned they never see half of the cases again and therefore cannot follow them to a conclusion.

DR. REED: I cannot give you a specific answer either. Many of these horses get away from us. Some fall down in class, through age barrier, and end up in Canada!

When selected properly, carpal fractures, with the exception of the slab fracture of the third carpal bone are extremely successful. How do you feel about the latter type of fracture, Dr. Wheat?

DR. WHEAT: In the last year, we have changed our procedure regarding fractures of the third carpal, so that I too, am in no position to quote figures. I do think that by removal of the slab we have had better results than by trying to anchor it in place with a bone screw.

DR. REED: That has been my impression too, for one specific reason. I think it is very, very difficult to get complete apposition and no have surface or cartilaginous erosion in the adjacent bone.

With regard to sesamoid fractures, again the answer is in doubt. Removal of a large fragment from the distal fourth of the bone, the basal fracture, entails considerable destruction and trouble may result, but in the case of the small, pinched-off, wedge-shaped fragment in the corner, the success rate is much better.

Getting back to the knee, the fragment off the distal end of

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the radius is easy to remove and merits a good prognosis. When a considerable surface area of the distal portion of the radial carpal is involved, the outlook is not as good. The reason for this is unknown, but may be architectural. Facts and figures, I cannot give you. I wish I could.

MODERATOR TEIGLAND: Next year we might entitle this panel "Improvement of the Breed in Canada"!

DR. JENNY: Many of us are murdering the carpus! In the case of an uncomplicated slab fracture of the third carpal bone, where on x-ray, a lateral exposure shows a distinct upward displacement while the horse is standing on the leg, but on flexion of the knee the same lateral exposure shows anatomic reduction, stabilization with a screw is the treatment of choice. Dr. Raker and I operate in this manner. We introduce a Kirschner wire, then take an x-ray to check the position of the wire. If apposition is good, the drill hole is made right alongside the wire. If the position is not ideal, we make the necessary correction while the wire is still in place. The screw is then inserted. The ones that really scarce me are those where damage to the main portion of th bone cannot be demonstrated even by radiography — and such cases do occur.

I wish to make it very clear that the uncomplicated slab fracture should be stabilized by a screw. After all, if one can make the approach without invading the joint capsule, good results should follow. We have radiographs of such cases, a year and more after surgery, which show absolutely no osteoarthritic changes.

I agree that we have become more cautious about sesamoid fractures. Again, it is a matter of selecting one's cases. If one encounters a lower border fracture and the fragment is well displaced, removal is indicated.

DR. C. H. REID: Mr. Chairman, I thought the question was rather terse, and perhaps I was not listening, but what was the final figure of the successful return to racing?

MODERATOR TEIGLAND: I interpreted that we would rather not talk about that! Dr. D. L. Proctor will now talk on "Post-surgical Management and Methods of Rehabalitation Following Bone and Joint Surgery".

DR. PROCTOR: I still don't like screws in a third carpal fracture. I am a real good student! Jacques Jenny showed me how to take them out, and it worked, and I am going to keep on taking them out!

When one starts out to apply human techniques to the horse, modification is necessary and the only way to become reasonably proficient is by constant repetition and evaluation of the results. The time for carpal surgery, in my case, has dropped from three hours to forty minutes.

It just happened that the first three cases of knee surgery I had an opportunity to work on were slab fractures of the third

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carpal. The first horse went on and won races. He re-injured that knee — the same type of fracture. The second horse had one of these displacements and flexion. He was the best friend I had in the east, winning eighteen or twenty races in New England. I don't think he was a sound horse, but he was economically sound — he won races. The third horse is starting his fourth year of racing, a horse called Lucky Me. He was second at Tropical Park last Monday and won a race on Wednesday.

My topic is "Postsurgical Care and Rehabilitation". Following carpal surgery, we put a cast on the leg. This is done to protect the incision but the horse ends up with areas of pressure necrosis over the area of the accessory carpal bone. This I regard as a calculated risk. As yet, I have never had a horse break open the cast or get down in the stall and harm himself. I am sure these animals would do just as well if one could get them to protect themselves without a cast.

I think it is wise to establish an antibiotic concentration in the horse prior to surgery, and to maintain it for at least ten days afterwards, because these pressure sores, due to the cast, are a constant sequel to knee surgery. I have tried everything I could think of, in the way of orthopedic felt, foam rubber. I finally hit on a sheared lamb's wool type of covering under the cast, to minimize the sores. I use Kymar ointment locally or Varidase and Kymar systemically, along with a topical application of Furacin solution or Urea Sulfanilamide alcohol. This preparation is put in a base of propylene glycol, which is similar to glycerin and has a sweating action as well as being bacteriostatic.

The cast is removed eight days later and the stitches usually on the second day after the removal of the cast. The reason we do not take the stitches out at the time we remove the cast is that edema occasionally develops following the cast removal. Naturally, we give the animal tetanus antitoxin.

We use 00 silk, with swedged-on needle. I think, when we do completely aseptic surgery and maintain a clean post-operative area by the use of the cast, we can leave the stitches in almost indefinitely. It is amazing how long these animals will tolerate 00 silk. Like everybody else, I like to get the stitches out, but I do not think it is absolutely essential to remove the stitches on any given day. If there is a minimum of tissue reaction, the silk can stay in for at least two weeks.

We found the animals were standing around on three legs, after surgery. This made a bad impression. The animals weren't eating, or feeling good, and the post-operative picture was not good. We have alleviated that to a great extent by very small doses of Butazolidin. We start them off on one gram, and maintain them on half a gram per day for four or five days after surgery, to get them over the incision soreness. I believe that is logical therapy. It has stood the test of time and certainly makes a much better

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impression on the owner, and I feel a lot better seeing a horse move freely about the stall, rather than standing there, anchored, on three legs.

If there is a certain amount of cellulitis, and the knee is larger than its companion, I like to use Pitman-Moore's Weladol in an attempt to sweat it and get it back as near to normal as possible without resorting to strong absorbents.

Another preparation that we use is a liniment. It is made up of 1 ounce of gum camphor, 1 ounce of menthol crystals, 1 ounce of oil of wintergreen, 8 ounces of tincture of arnica, and alcohol to make a quart. This preparation does not blister; it tightens and has an absorbent effect without the disadvantage of causing a skin reaction.

We do not exercise them for the first ten days after surgery. As soon as the cast is off, we walk them for half an hour a day. Bearing in mind that this is a typical stiff-kneed gait, it is well to have adequate bedding in the stall. A knee-deep bed is unnecessary. This horse cannot get the cast up over a heavily bedded stall.

At this point, some fourteen to twenty-one days post-surgery, radioactive cobalt comes into play. We routinely radiate each of the joints with 1000 r. At six weeks a post-operative x-ray is taken and if there is indication of fibrosis or commencing calcification, we don't hesitate to repeat the 1000 r.

In the post-operative handling of sesamoid fractures, we do not use a cast. We simply use an Ace bandage, and nothing other than that, and we start walking them the day after surgery. Other than that, the aftercare is the same as for carpal surgery.

I want to tell you gentlemen that, if you listened carefully to what Dr. Wheat said a moment ago and had I had the privilege of listening carefully to him some six months ago, I would have escaped a very embarrassing situation.

I was called to Springfield, Illinois, to look at a trotting filly that had an apical fracture of sesamoid on the hind leg. It was a very clear fracture with no displacement.

With the very best intention in the world and thinking we knew exactly what we were talking about, we told the gentleman that this filly was a subject for surgery, or if surgery wasn't desired at least she should be kept in a stall, a light cast applied, and given complete stall rest for a period of six weeks. The man decided to make a brood mare out of her. He took her home but couldn't stand seeing her in the stall. She was kicking and playing. He turned her out in a small paddock where she put on a regular circus act in the paddock. He called me and said, "Dr. Proctor, this filly is not lame under exercise in the paddock".

I replied that in all probability she could be sound in pasture, but that if the filly was put to work, separation of the sesamoid and possibly suspensory breakdown would occur. I told him to put her back in the stall and keep her quiet.

So, he turned her out in the pasture! She was still sound at the end of the week. He put her back in racing. In August, this filly ran in the Illinois Filly Stakes ($20,000) and won the stake at the Illinois State Fair. I saw her and actually drove her myself at Lexington in September. She is still sound. I have re-x-rayed her. The fracture is still here.

So, as I said, had I had the privilege of sitting here and listening to Dr. Wheat in July, I would have paid several times the price of the trip to Phoenix to avoid that embarrassment.

This is by way of telling you that in a case of complete fracture of the sesamoid, if it doesn't involve any displacement, not only is surgery not indicated but I am not sure you should take them out of training!

Another point that I have had brought home to me very vividly, and much to my embarrassment and concern, is the fact that, even though we had tremendously clear x-rays from every conceivable view, I have never yet gone into a joint but what I have been dismayed to find that the pathology is considerably worse than the x-ray would indicate. If it looks bad on the x-ray, gentlemen, when you get in there, it will terrify you!

Another little tip I learned the hard way, or a thought for you to bear in mind, is that if you aspirate a joint (probably contemplating the use of corticosteroids), and find that the joint fluid is blood-tinged, you had better use your persuasive powers and get the owner or trainer to give you permission to do a thorough x-ray examination. You will find that 70 to 80 per cent of these blood-tinged synovial joints have some type of fracture. I am thinking now particularly of the carpus. If there is blood-tinged synovia, there is probably a small chip somewhere. I don't think these little chips are responsible for many of the clinical syndromes, and I would like to emphasize that I don't think any of these conditions are ever static; they are either getting better or worse. For instance, if fibrosis is beginning to be dense enough to show a lightened area on a radiograph, I feel it is just a question of time till it actually calcifies. If for no other reason, removal is indicated.

Dr. Wheat showed a picture of tremendous calcification on the proximal end of the metacarpus and over the third carpal bone. This was Nature's attempt to heal a slab fracture of the third carpal.

I would like to show you a couple of slides of pathology that was amenable to surgery. These pictures were taken six- and twelve-weeks' postsurgery and show that the tremendous amount of calcification does not necessarily come back. All of these animals I show today have responded to carpal surgery and have been economic successes.

[Slide] This is a chip off of the radial carpal, and the resulting calcification is Nature's attempt to bind that sequestrum

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in place. The whole thing originated from a small sequestrum, not much bigger than the eraser at the head of an ordinary lead pencil. This mare had only about 15 per cent flexion, and couldn't even take care of herself among the other brood mares in the pasture.

We operated in an attempt to open up the knee and make this mare pasture-sound. She responded so well, she kicked the side out of a training ring. This mare won three races. A good bit of the calcium did return, but enough money was made by the mare to pay her training bill for the year and stud fee. She is now back in the brood mare pasture and in foal.

[Slide] This is that same joint, 120 days after surgery. We went in there with a curette and cleaned the joint out, trying not to interfere with the articular surfaces. We took out everything that was abnormal. Despite Dr. Jenny's assertion that there is no reason to suppose that that calicum on the front end of the radial carpal would necessarily cause any pain, it is a terrible blemish, and just as easy to take it out as to leave it in. So we took it out. It certainly looks a lot better.

[Slide] This is of the horse Lucky Me, and the type of slab fracture with displacement we were speaking of. We simply removed that. This horse, as I say, is entering his fourth year of racing and is certainly an economic success.

[Slide] This is postoperative on the same horse. I didn't get as good a view as I should. I didn't get the right angle. The significance of this picture is that the joint itself is clean, and there is apparently no calcification following the removal.

[Slide] This is a filly that belonged to the artist, Allen Brewer. There was a tremendous amount of calcium. This, too, originated from a chip off the radial carpal. This particular filly won four races, was four times second and out of the money a couple of times. The condition has recurred. He got one short racing season out of her but he picked up several checks, and she certainly paid her way. She, too, is back as a brood mare.

[Slide] This is one of those types that Dr. Reed spoke of as being very amenable to surgery.

This chip was off the radial carpal bone. You can see the stainless steel sutures I bury. This slide was taken some 120 days after surgery. There is a good, clean knee, and no evidence of calcification. This animal and other animals were irradiated, by the way.

[Slide] This is the horse called Mr. Vale who was a Kentucky Derby candidate. This was a chip off the posterior end of the distal and lateral surface. This is the postoperative picture. This particular animal went on and raced successfully. We have given you the ones that have done well. There were several of them that did not do well. I raised the question of percentage, not maliciously but simply because I wanted to know. It is hard to keep track of

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these animals for they go all over the country. Since the other members of the panel are not dogmatic, and I am seldom right but never in doubt, I will give you what I think is a fair approximation.

Our carpal surgery is over 70 per cent successful. I feel that our basal sesamoid surgery is between 25 to 30 per cent. I think you should remember that if there is any distortion or clubfoot type of calcification with a subluxation of the first phalanx, the prognosis has to be exceptionally guarded. I agree with Dr. Wheat that surgery is probably not indicated in the basal fracture characterized by distortion of the physiological angle of the phalanges.

I believe the outlook for apical sesamoid fractures is twice as good as for basal fractures. There, again, each case is an entity, and the size of the fracture enters the discussion. I would say 25 to 30 per cent of the basal fractures go back to work and probably over 60 per cent of the apical fractures of the sesamoid.

In a series of 16 sesamoid operations, 10 fractures involved the right leg, 6 were on the left leg. Seven of them were apical, and nine were basal. Eleven involved the medial and five involved the lateral sesamoid. I do not think there is anything very significant about this. It just shows that sesamoid fractures happen on both the right and the left legs, and medial and lateral sesamoid fractures occur in front or behind.

A different "cup of tea" on carpal injuries has come to my attention. In a series of 27 knees presented for surgery, 22 involved the right knee and 5 the left knee. Sixteen of the 27 involved the radial carpal, and 5 involved the third carpal; six involved other portions of the carpus or were so extensive that multiple areas were involved.

What this thing boils down to is that you can almost know, by the time you look at a knee, that there is a particular part of the carpus involved, and it is usually the inner distal border of the radial carpal bone. Why it is the right knee, I cannot tell, but time after time it involves a sequestrum of the radial carpal on the right leg.

MODERATOR TEIGLAND: We are now open for questions for Dr. Proctor.

DR. DELAHANTY: This question is directed to Dr. Jenny. Will we get more periosteal reaction from early moderate ambulation, by this I mean walking from the first day of surgery onwards or should we wait several weeks until we get organization? What is the criterion for the decision of exercise or no exercise immediately after surgery? I refer particularly to carpal not sesamoid factures.

DR. JENNY: I cannot give a specific answer, but I am sure it makes good sense not to exercise a horse immediately after invasion of the carpal joint. If one makes a comparison with human

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surgery, nobody pushes an old person out of bed after a meniscus injury had been operated upon. Maybe the horse loses even more joint fluid. One thing is sure, post-surgical joint fluid is of inferior quality. At what time it returns to normal is not know. I think it would be very well worthwhile to investigate this problem.

DR. PROCTOR: As I said, we put our carpal cases in casts and there is no way I can walk them successfully, but I think I would probably try to do it if I could! I am not at all sure but I see no reason why such cases should not be exercised a little, provided they are not dead lame. The horse's foot is dependent on movement for proper circulation. I believe that exercise increases the normal circulation to the leg. Limited exercise following surgery is a good thing.

DR. RILEY: I have a question for Dr. Jenny. Is there anything in the literature about joint fluid being withdrawn into a sterile container, prior to surgery and then being reinjected into the joint immediately following closure — assuming the joint was not infected?

DR. JENNY: Van Pelt in Michigan and Link in Illinois, I believe did just that but most of the work I am familiar with stems from Swedish literature and from Johnson's work and I cannot add much to that which I mentioned in my paper.

DR. POWELL: I would like to ask Dr. Proctor about his nutritional schedule for these horses after surgery and up to the time they are put back into training.

DR. PROCTOR: I do not make any special attempt to provide high calcium or phosphorus intake. The animal is kept on a diet of the best mixed hay I can afford to buy and top-quality oats. We use some sweet feed and Jen-Sal's Vimogen is the mineral supplement of my choice. Other than that, they are fed like any other animal, according to their individual condition and appetite.

MODERATOR TEIGLAND: The last speaker on the panel is Dr. W. O. Reed who will talk about "Humane Aspects of Bone and Joint Injuries and Initial Therapy on the Track or Farm".

DR. REED: Before going ahead with the topic assigned to me, and in regard to matters already discussed, I would just like to stress that I think it is wrong for us to stand up here and say to you that these things are simple. They might be simple to those of us who have all the necessary equipment but it is not simple if you do not have the facilities or equipment. It is not simple if you do not know how to select the cases and it is not simple if you do not have adequate post-operative facilities. For instance, one of the important considerations is the use of post-operative radiation. I also believe that where the necessary facilities are not available, it might be advisable, after selecting the case liable to profit from surgery, to refer it to some one who not

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only is equipped for this type of work but who is also actively engaged in such work.

I will discuss, briefly, the second part of my topic before dealing with the more controversial humane aspect. I am referring primarily to fractures or injuries of almost any kind that occur on the track or farm. We first attempt to adequately tranquilize the animal. Often the shock of an accident and the inability of the animal to cope with a completely new situation causes a somewhat hysterical attitude on the part of the horse. This is evidenced by the fact that on many occasions a very simple fracture readily becomes a comminuted fracture if the horse is not tranquilized. Sparine or Promazine is our tranquilizer of choice.

Control of pain is a factor, however I do not believe it is as great a factor as we think. For a period of roughly half an hour after injury, the area is usually numb. Plain in a properly immobilized fracture is usually not extreme even after the return of sensation. The relief of pain is, however materially assisted by the use of Demerol, Dyprone, etc. Our preference is Demerol. A drug of assistance in the control of muscle spasm is Robaxin. These two considerations, tranquilization and the relief of pain and spasm will often prevent needless future self-destruction on the part of the patient.

The next and perhaps most important consideration is the immobilization of the fracture area. It is my opinion that this should be done as soon as possible. Many times, swelling in a joint can be prevented by the immediate application of a plaster of Paris cast. Our preference is Velrock in conjunction with stockinette and where necessary to prevent skin irritation, foam rubber. In recent years, the thinking of human orthopedists has changed greatly from the old idea of allowing swelling to reduce before applying a cast, to the newer procedure of applying a cast even when swelling is present. You may believe that since the area is so greatly swollen, a cast is contra-indicated, but if one is applied as soon as possible after injury much of the swelling will not occur. If such a cast becomes loose, a fresh one is put on. I put on as many as five new casts in a period of a week.

For some years, Dr. Jenny has been working on an emergency splint which can be applied immediately following injury and thus enable the horse to be moved from the track or pasture to permanent stabling. He has, as chairman of our Committee on Bone and Joint Injuries, submitted to the Grayson Foundation a request for assistance with this project. I have seen this equipment and it offers an excellent means of removing an injured animal from the race track. Race-track management attempts to go all out to satisfy the betting public. It is quite certain that spectators would much prefer to see a first-aid crew go into action than see somebody come out and shoot the horse.

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Some years ago at the National Horse Show at Madison Square Garden, a horse, going over a jump, fractured a leg. Fully three-quarters of an hour were spent in hysterical manipulation before the house lights were lowered and the animal shot in the presence of several thousand people. The public reaction was understandable and the press publicity was enormous.

This year, the Derby favorite in England, a French horse named Angers, broke a leg and was destroyed on the track. Some very controversial comments regarding that situation were printed in the Veterinary Record, the official publication of the British Veterinary Association.

On many occasions, even veterinarians are guilty of hysteria, or at least lack of judgment following injuries to horses on the track. There is a great tendency for the track veterinarian and examining veterinarian to immediately want to destroy a horse. To me, even though the accident may have had serious results, it is just impossible to properly evaluate a case without proper x-ray examination and careful consideration of all the facts. I grant, that if a horse's leg is dangling loosely, that is another matter, but few cases are as obvious as this. There have been cases where a track veterinarian ordered a horse removed in an ambulance and to be destroyed, but the stable veterinarian happened to come by and requested a more thorough examination. One such case involved a horse which later won some $50,000!

It is my opinion that a standing rule or consideration be made that at no time should the track veterinarian destroy a horse without consulting the stable veterinarian. This is not to infer that the track veterinarian is not competent, but previous experience has shown that many horses are destroyed needlessly.

It is also our opinion that if proper facilities, such as first-aid measures and a horse ambulance are available, no horse should be destroyed in full view of the public at the race track. Imagine some forty or fifty thousand spectators leaving a race track with various degrees of condemnation of the veterinary profession because they witnessed the public destruction of a racehorse. If a horse must be destroyed, it should not be a public spectacle nor should it be carried out behind a cheesecloth screen.

The humane aspects of bone and joint injuries have been left to the end of this topic because of the controversial aspect. I refer primarily to the position into which we are forced by the insurance company and the client. Unfortunately, once again the veterinary profession is undeservedly very much on the spot, for in many cases the decision to destroy or salvage the horse depends on whether the horse was insured or the amount of insurance involved. Insurance companies are more lenient toward destruction when a horse is insured for a small amount rather than for a large sum. This, I consider to be most unfortunate.

At present, there is an absence of standards, with the exception of those established in the minds of some more experienced practitioners. Perhaps a great deal of criticism of this rather hopeless situation should be directed at the agent who, in selling insurance to the horseman, infers that he can collect on almost anything. It is obvious that, with newer surgical technics, the successes of many cases will predicate against destruction of cases which in the past have been routinely destroyed. This is daily becoming more and more obvious.

We must bear in mind the ultimate outcome of the case — not just the immediate question of severity of pain — including the matter of the horse being able to lead a useful life. The Archbishop of Canterbury recently criticized surgery on aged persons merely to continue life for a very short period of time. It is our opinion that this same philosophy should apply the horses. I cannot see the justification for keeping a horse alive if it does not have a good, normal, useful life. This, of course, does not mean that it must return to racing soundness. In this connection, however, the use to which the animal is to be put must be kept in mind, for it would be foolish to perform extensive bone surgery on an aged gelding. The environment to which the animal must be exposed and his disposition must also be considered.

In conclusion, I feel that at no time should there be a guilt complex on the part of the veterinarian as the result of undue pressure either from the client or insurance company. I believe that we should have certain standards for those cases whether they are considered reparable or irreparable.

MODERATOR TEIGLAND: Before we conclude this panel, has anybody got any further questions or comments?

DR. JENNY: I have been asked to make a few comments on the activities of the committee on bone and joint injuries. We prepared an outline or proposal for the Grayson Foundation in which the ideas resulting from our discussion last year were incorporated. Dr. Kester has summarized my paper and I should like to read this summary to you:

"I. **Facts bearing on the problem:**

1. Orthopedic surgeons in human medicine have reached a high degree of proficiency in handling bone fracture cases in man.

2. Veterinary orthopedic surgeons have reached a comparable high level of proficiency in handling bone fracture cases in small animals.

3. The reason for the foregoing is the fact that both medical and small animal veterinary orthopedic surgeons are required to treat and handle vast numbers of cases.

4. Orthopedic surgery and the handling of fracture cases in the horse **could** but **has not** reached the same high level of pro-

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ficiency. The reason is that horse owners seldom allow fracture cases to be treated. Thus there has been little opportunity for veterinary surgeons to develop proficiency in handling fracture cases in the equine.

II. **The problem** is to obtain more naturally occurring fracture cases for equine orthopedic surgeons to treat so that proficiency may be developed through experience.

III. **Obstacles** to overcome:

1. Public opinion. The public can be assured that it is not cruel to treat a horse with a fracture and would be quick to grasp this improved humane approach.

2. Competent first-aid facilities and treatment would be required at co-operating race tracks. Existing facilities can be readily adapted with little difficulty and cost.

3. The technical problem of handling and moving the injured horse. Proficiency needs to be developed in this area. However, pilot runs indicate this is not a serious problem and should in no way serve as a deterrent.

4. Obtaining the patients. Some means must be devised whereby the owner can dispose of and receive compensation for the horse with a fractured leg without first destroying the animal. A satisfactory arrangement very advantageous to both the owner and the insurance companies could be worked out.

IV. **Solution to the problem.**

1. Establish an equine orthopedic center at one of our veterinary colleges where large numbers of cases can be handled.

2. Obtain or establish a Foundation or other agency for the purpose of organizing and implementing the program which is suggested by the above facts and which is described in detail in the attached proposal.

V. **Comments.** The proposal is entirely practical, desirable, and should be implemented at once. It is directly in line with the thinking and action of the American Association of Equine Practitioners at their last annual meeting. We sincerely hope that insurance companies, horse owners and related agencies and interests outside the veterinary profession will also give the proposal their whole-hearted support."

I strongly urge you to support our effort. I would like to make an additional comment about the controversy in the British press regarding the "Angers" case. A colleague wrote in "The Veterinary Record" — "If proper facilities are not available at race tracks, members of the profession should decline to function." If we all put some pressure on track officials before we have an "Angers" tragedy, we might save ourselves a lot of embarrassment.

DR. O'DEA: In regard to the Horse Show Committee, this is

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a question that might come up and involve men who are serving at horse shows. After that accident in Madison Square Garden, the situation appeared to be a lack of organization, where every professional horseman got in the act and the place became a madhouse. The veterinary situation was a divided one. They had an attending veterinarian and an official veterinarian. The official veterinarian gave the decisions in the ring. Nobody knew whose position was official. We decided to put everything under one head. We organized the police force at the Garden so that if anything happened nobody was to be allowed in the ring except those officially entitled to be there. When you people take part in the small shows and an accident occurs, be sure that nobody except the owner and the veterinarian gets into the ring. You need the owner and perhaps the handler of the horse. It is advisable to have a pail of water and maybe six bandages. You can put on a wet bandage to support the injured leg before trying to move the horse. This is simply first aid. We have had it happen in Madison Square Garden where we have been able to support the horse so that we could load him and we have not had any displacement or compounding of the fracture as they formerly had.

If destruction is mandatory and the horse cannot be got out of the ring, use Lethol or something similar. Inject it into the jugular vein through 18 or 20 gauge needle. It works beautifully even in an excited horse. We use a 100 cc syringe and a 12 gauge needle.

DR. WISEMAN: I have talked to Dr. Jenny and I understand he has a temporary splint. An old Mexican taught me how to make an emergency splint. He took a piece of wide-bore black plastic pipe and split it down the middle. Then two clamps, similar to those used on a radiator hose (you can get them in different sizes) are used to clamp the splint in place over a temporary cast. Bandage material is applied underneath the cast to make the surface level.

DR. NICHOLS: I would like to ask if cortisone or Furacin is indicated in joint surgery.

DR. JENNY: Dr. Nichols, I would say neither. First of all, the only local antibiotic we use is crystalline penicillin and streptomycin. We like to make sure, before we close the joint, that we do not have any blood clots in it. A blood cot is an avascular clump of tissues which can harbour bacteria out of reach of systemic antibiotics. We try to practice as strict asepsis as possible and then depend upon an antibiotic umbrella for about 5-8 days postoperatively.

Cortisone I consider definitely contra-indicated post-operatively. While you effectively depress inflammation you also depress tissue repair. Furthermore you lower the resistance to infection.

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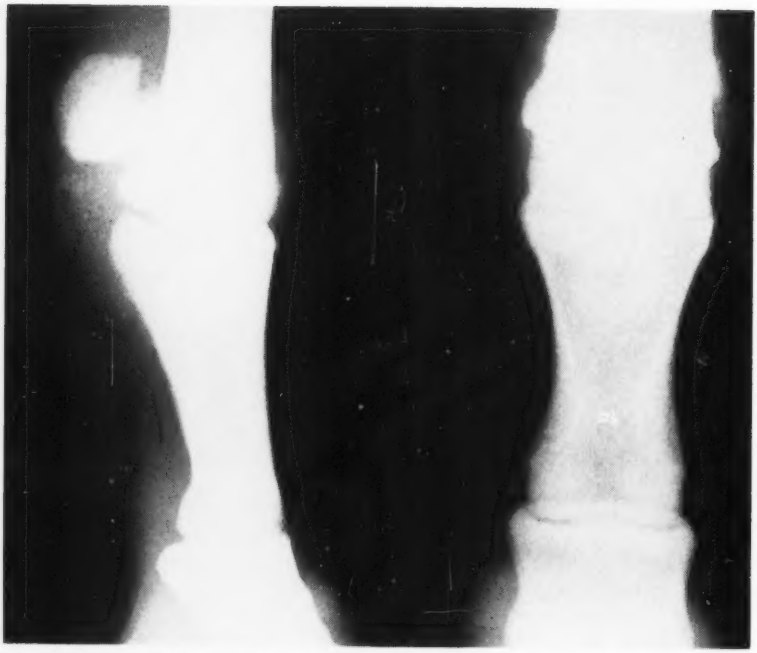
J. W. WHEAT. Fig. 1. Carpal exostosis with obscuring of the fracture line.

J. D. WHEAT. Fig. 2. Sesamoid fracture with excessive size of fragment.



J. D. WHEAT. Fig. 3. Dorsal luxation of the sesamoid bone.

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DO'S AND DON'T'S OF PRACTICE

CHAIRMAN TEIGLAND: In this part of the program, I am going to call on individual members of our association to come up to the platform and tell us some do's and don't's of practice.

Every day in general practice, little incidents occur which might end more happily if we had been aware of how the other man overcomes the situation. Perhaps some of our members have some new ideas or gadgets to bring to our notice.

Dr. Simensen from New Hampshire, will you start the ball rolling?

DR. SIMENSEN: Before using a new line firing iron, it is advisable to dull the edge with a file, because, when new, red-hot and sharp, it can get one into a lot of trouble.

In giving a bolus to a horse, it helps to wash out the mouth with a dose-syringe full of water. This gets rid of the residual hay and facilitates passage of the bolus. Incidentally, from Dr. Burch, I learned to use a 13-inch balling gun instead of the 16-inch model.

I also prefer to take about four inches out of the middle of the long dental float. It makes for easier rasping.

To protect cuts on the face and around the eyes, I bought some plastic dish pans and cut them to fit the side of the face. They can be fastened to the halter and are very effective in preventing the horse from rubbing the lesion.

Before using a new emasculator, the sharp edge should be dulled by cutting on a piece of rope.

In neurectomy, by pulling on the nerve a little more than is customary before sectioning, the proximal end will retract and thus not adhere to the healed incision site.

When using a bar shoe for a fractured coffin bone, ensure that the blacksmith does not make the junction of bar and shoe right under the fracture line.

When giving piperazine powder in the feed, put a pinch of the powder into the horse's nostril before mixing the powder with the feed in the tub.

Finally, when dispensing liquid for a high level dose, dispense in small amounts so that the over-zealous groom who thinks that if a little is good, a lot is much better, won't get you into trouble.

EDITOR'S NOTE: At this point, Dr. Courtwright demonstrated a speculum-culture-tube carrier which can be autoclaved. It was designed fifteen years ago by Dr. Courtwright and has used only two or three light bulbs in that period. (Fig. 1).

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DR. LINFOOT. Horses which are stabled farthest from the track show a lower incidence of typing-up. I think this lower incidence is due to the warming-up period before strenous exertion. I have therefore made it a practice, to have the horses walked out for about fifteen minutes as a warming-up period prior to exertion. This, along with medication such as laxatives, tranquilizers, especially in the more nervous individuals and multiple vitamin therapy has lowered the incidence of this condition.

My second observation concerns the many ways or variations in which the influenza complex shows itself. For instance, in the remount station, I noticed that many recovered horses would show joint involvement. In the paddocks, horses would be noticed with an upward fixation of the patella. We would reduce the subluxation but it would recur. I formerly believed that these were chronic cases but now I think that most of these will correct themselves spontaneously if given time. Adequate convalescence is necessary to prevent the bowing of tendons and breakdowns which may occur soon after an influenza attack if the horse is returned to work too soon.

DR. HUMPHREY: The collection of blood for transfusion purposes can at times be a frustrating procedure especially if vacuum bottles are not available. To overcome this deficiency, I use a 50 cc. syringe along with a B. D. adapter, a rubber cork, two glass rods and a gallon bottle. With this equipment, I can collect a gallon of whole blood, froth-free, in five minutes by using a bleeding trocar or large gauge needle. The equipment can be sterilized and it is easy to carry around. Transfusion, instead of being a one or two hour procedure can be rapidly carried out.

CHAIRMAN TEIGLAND: If any of you are embarrassed to tell about your mistakes, I shall tell you one of mine.

I had occasion to fire both ankles of a horse. The assistant was applying the paint to the left ankle when I noticed I had missed a number of punctures on the inside of one of the legs, so I sat down and started firing again just after the leg paint had been applied. This is something like a "shishkabob"! You do a real job of firing! Fortunately, a towel wrapped quickly around the leg takes care of everything, but this could be as dangerous as a fire in a stall.

DR. H. M. SMITH: I was particularly impressed by Dr. Reed's comments on humane destruction of animals. If one picks up Medical Economics or any similar publication, it is noted that the medical profession is being subjected to an increasing number of malpractice suits. A lot of this may start to rub off on to the veterinary profession. Most of us have probably been fortunate in this regard but in officiating at hunt meets, race tracks and horse

shows, when the occasion arises that an animal has to be destroyed, I would caution everyone — and I hope that this will be incorporated in the recommendations of the Horse Show Committee — to ask either the owner of the horse, or his agent, to sign a release permitting you to destroy the horse. If you are under pressure and in a hurry, pull out your prescription pad and say, "Make out a release and sign it; I shall fill in the details later". I believe this is very important from a medico-legal standpoint, otherwise you may find yourself being sued for $5,000 on account of a $250 skate.

In his paper, Dr. Reid of California mentioned a case of chondroids in the guttural pouch. At school, we had lots of practice on cadavers in passing a drainage catheter into the pouch, but unless one is doing this constantly, proficiency is lost. For about fifteen years I did not see a single case of empyema of the guttural pouch but in an eighteen month period, five or six cases were encountered following strangles. I drain the pouch as follows: The canula from a Kentucky horse trocar is replaced by a piece of 3/32 polyethylene tubing (disposable intravenous tubing) or instead of a trocar a 3/32 or 5/32 intramedullary pin may be used. Following local anesthesia, an incision is made through the skin of Viborg's triangle. The instrument is then passed alongside the larynx and into the bottom of the pouch and the trocar removed. The modified canula can then be held in place by suturing to the skin. Daily irrigation with enzymes and antibiotics can now be carried out. Within four or five days the thick pus is liquified and uneventful recovery results.

My third comment is with reference to intravenous fluid therapy. It can be a backbreaking and tedious task to stand and hold a flask while an amount of fluid varying from four to twenty liters is being administered. The use of a dumb jock as used in breaking colts is a handy apparatus to overcome the difficulty. A six foot long bar of ¼" x 1° aluminum is bent into a semi-lyre shape. Four holes are drilled in the bar. One hole is drilled about two inches from each end of the bar and the other two holes are drilled on either side of the middle of the bar and correspond to the holes in the saddle into which the rings or terrets are screwed. The terrets are used to hold the apparatus in place (Fig. 2). Intravenous flasks can be attached and with the horse on a crosstie, administration can be accomplished without difficulty.

DR. C. H. REID: First of all I would like to say that I am of the same opinion as I had last year. I get more out of this meeting than any I have ever attended. This particular group comes up with a lot of good professional meat and I always go home feeling that I am super-educated.

I have been asked some questions about the motion picture

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stills that you saw on the easel and I have picked out two of them to show you again. This particular shot was made in the picture "Vera Cruz" in Mexico. The horse is jumping across a canyon 28 feet wide. His hind feet are already four feet lower than the take-off point and the difference in height between the two sides of the canyon is 18 feet. The stunt man who made the jump got $500 for his effort and after he had done it, he looked back at the canyon, broke out in a cold sweat and said "I would not do it again for $10,000".

The second picture is of a black bear weighing about 600 lbs. I was called to see this bear because he had a rather dangerous habit while on the set, of rushing up without warning and grabbing a person across the thigh. If you have ever looked at a bear's tusks you can figure how easily they can get down to the femoral vein. It is a good thing to have your sneakers on at all times!

I worked on this bear about eight or nine years ago and went to see him about a week ago, not having seen him in the meantime. The man who owns him said, "He sure remembers you. Let's get out of here". I wanted another picture of that bear but did not get it. Whenever these animals start making a noise like a pig rooting in the dirt, get out fast. This particular bear put many people in hospital, but since it was such a valuable animal for motion picture purposes, the owned decided to cut off his tusks. I went with him into the cage, which was no mean feat at that time, but I had more nerve then than I have now. I gave him morphine and got him pretty drunk, put on a rope and handed it to two fellows outside the cage. In a drunken stupor, unable to raise his head off the floor, he grunted and those two fellows slid across the dry ground like holding on to nothing! That gives you an idea of the power of these animals even when under narcosis. I took a little motor tool and cut off the tusks. My dentist had supplied me with the necessary filling so I dug out as much nerve as possible and filled the cavities with silver filling. By now the bear was lying so peacefully that I said to the owner, "You know, John, I don't think that what we have just done is going to take it out of his head. Why don't we castrate him at the same time?" "Have you ever castrated a bear?" he replied. "No! but I will never have a better opportunity"! As far as I know, it did make some difference to the bear, but not enough to say he is now a safe animal. To my knowledge he is the only castrated bear with silver fillings in his teeth!

DR. POWELL: I have been using a twitch which is both simple and inexpensive. A figure of eight is made out of $\frac{3}{8}$" hot rolled steel. The two loops are of unequal diameter. The small loop to which the nylon loop is attached has an inside diameter of $1\frac{1}{2}$" and the large loop an internal diameter of 6". A twitch

of this size can be put on from the side and in addition it can be tied or snapped into the side of the halter leaving the attendant free to hold the shank. If the horse becomes violent, you can stand back holding the shank.

The second thing we have developed to help us in back-yard practice is a twelve foot length of ½" cotton rope into one end of which is woven a ring made of ½" iron rod with an inside diameter of 2". To apply this improvised halter, you ease up to the horse's neck, drop the ring over the far side of the neck. Take a loop of the rope and pass it through the ring and over the horse's nose. To turn the horse loose, drop the loop off the nose, pull the rope and it falls away.

With regard to lawsuits for malpractice, collection of fees, etc. I find it very valuable to carry in the car one of those little triplicating machines in which all details of the case are entered. Entries are made at the time of the visit. A copy is given to the owner if he is present, the other two are returned to the office. My secretary mails one out at the end of the month and the original is retained. These records are accepted in court as evidence of the call.

I would like to endorse the value of Dr. Courtwright's swabbing instrument. I believe that if you look around as I did, you will find an old bronchoscope going cheaply and with very little adaptation they can be used for taking swabs.

CHAIRMAN TEIGLAND: Regarding insurance, I do not think that the A.V.M.A. policy gives enough coverage. If I remember, the limits are five thousand dollars for any one case and a total of fifteen thousand for an aggregate of three cases per year. We have recently taken out a policy with a company which handles all our liability with coverage of one hundred thousand and three hundred thousand dollars, and it does not cost very much more than the A.V.M.A. policy.

DR. KESTER: Don't confuse rattlesnake bite with strangles. When the snake bites the horse on the nose, a terrific cellulitis results. If the horse survives, a nasal discharge is seen on the second or third day, but what upsets the horse more than anything is the swelling of the upper respiratory passage. He will suffocate. Another problem is what one might call the "downhill disease". The horse looks as if it might be developing azoturia or colic. Perhaps you learn that the horse has been running uphill or on the level for the previous several years. All at once he goes up and downhill and must feel as we do if we have been running up and downstairs. If you are in doubt about the diagnosis, run him uphill and he will recover instantly. The most classical case I have seen was when the horse almost fell down when being

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jogged downhill. He was turned uphill and no one could tell that there was anything wrong. All that horse required was twenty-four hours rest. You meet these cases on trail rides and in pleasure horses.

DR. C. H. REID: Ninety per cent of the time a horse struck by rattlesnake gets bitten on the nose. A tracheotomy tube is essential for I have seen the nostrils close completely. Do not be afraid to puncture the swelling. I make as many as 25 or 30 punctures with a nicking knife and the swelling will recede much quicker than it will by any form of medication.

DR. CARROLL: We see a few snake bite cases. We take a piece of plastic hose about six or eight inches long and place it up the nostril, tying the end of the tube to the halter. The swelling will close down on the tube but the horse will still be able to breathe.

DR. BURGER: For about eight years we have used cortisone for treating snake bites. The results have been very dramatic, so that in the snake season we leave cortisone on the ranches which are twenty or thiry miles away. We have worked ourselves out of business but it is very gratifying to see the horses recover so dramatically.

DR. A. M. SMITH: I don't use antihistamines for snake bite cases. I believe that research has shown this agent to be contraindicated in this condition.

DR. E. R. WALKER: To destroy horses we use an ordinary solution of Roccal. Eighty to one hundred cc injected rapidly will destroy an adult horse or cow.

DR. WISEMAN: Straight ten per cent?

DR. WALKER: Yes, just as it comes in the bottle.

CHAIRMAN TEIGLAND: I use fifty percent. It is very thick.

DR. OWEN: A modification of the one-man twitch mentioned by Dr. Powell is to use a double snap. Put the nylon loop in one end of the snap, twist up and fix the other end of the snap to the halter. (Fig. 3).

DR. COURTWRIGHT: I have on occasions run into trouble trying to give intravenous fluids or blood to young foals. If you hold them, they fuss and in moving their heads they ultimately dislodge the needle. To overcome this, I take a piece of heavy bone wire about 2½ inches long. At its middle, it is bent around the hub of the needle, something like a hairpin, and the two arms of the wire are bent at right angles so that they run parallel with

the shaft of the needle. The free ends are then bent at right angles away from the needle. When the needle is in the vein, two Michel clips are placed over the last bend and these retain the wire and needle in place. I even use it on occasions in adult horses when giving prolonged drips.

DR. WISEMAN: I have been asked about the temporary splint made from irrigation pipe. The diameter of the pipe is four or five inches and the thickness of the wall is less than one quarter of an inch. In summertime it is pliable. The pipe is split lengthwise and put around the leg after the area is built up with bandages. The clamps are then applied.

DR. TEMPLE: If in passing a stomach tube, difficulty is encountered in getting it past the epiglottis, use a large size of tube. If the tube becomes dry and sticky once in the esophagus, blow into the tube and thus create an air pocket through which the tube will pass, thus obviating the necessity for pulling out the tube and oiling it again. One other item about collecting that bill! If I don't get anything out of the trainer after thirty days, I go to the owner and if he does not come through within thirty days I really jump on him. Even though they may scream and holler, the end result is gratifying. I got so disgusted with one man that I went to the stewards about him — and now he is one of my best clients! If you allow a bad payer to get away with it, you end up by losing the stable, the owner, time, money, good humor, self respect and everything that goes with it!

CHAIRMAN TEIGLAND: At this time I would like to thank all those responsible for this program. You have no idea how difficult it is to have a program of this type unless you have the type of men who will participate. Their efforts are greatly appreciated. The Local Committee has done an outstanding job and I thank everyone who participated in the program.

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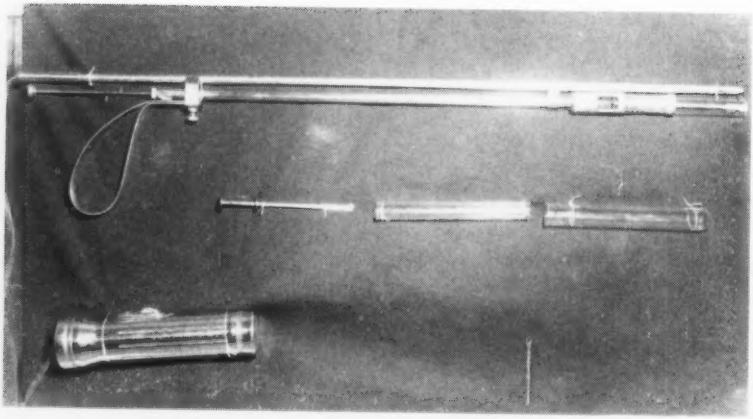

COURTWRIGHT. Fig. 1. Speculum-culture tube carrier.

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SMITH. Fig. 2. A dumb jock for intravenous infusion.

OWEN. Fig. 3. A modified one-man twitch.

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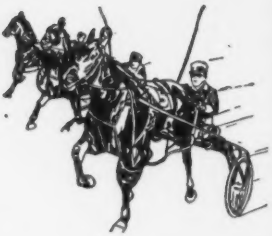

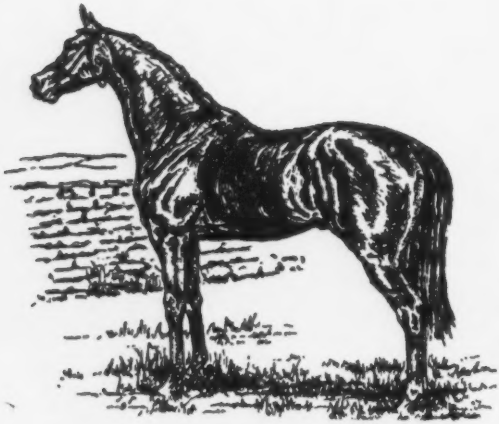

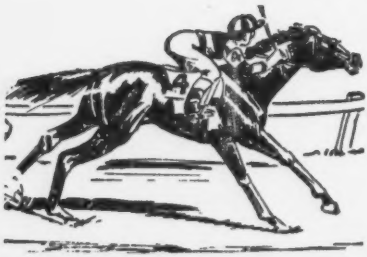

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